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Environmental Pathway Models for Estimating Population Health Effects from Disposal of High-Level Radioactive Waste in Geologic Repositories. Final Report

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16. ABSTRACT

As part of its program to develop environmental standards for disposal of high-level radioactive wastes, the Environmental Protection Agency estimated population health risks for 10,000 years after disposal in mined geologic repositories (EPA85a). This report describes the mathematical models EPA formulated to calculate the environmental risk committments (ERC's ---fatal cancers and serious genetic effects to all generations) that could occur as a result of releases from such repositories. The report also identifies the data EPA chose to use in these models and presents our estimates of the premature fatal cancers caused per unit of radionuclide released to the accessible environment. These estimates have been used to select the containment requirements in the Agency's final disposal standards (40CFR191).

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ENVIRONMENTAL PATHWAY MODELS FOR ESTIMATING POPULATION HEALTH EFFECTS FROM DISPOSAL OF HIGH-LEVEL RADIOACTIVE WASTE IN GEOLOGIC REPOSITORIES

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Final Report

August 1985

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FOREWORD

The Agency published environmental standards addressing disposal of high-level radioactive wastes (40 CFR 191) for public review and comment in December 1982 (47 FR 58196). The comments received have been considered and the Agency has promulgated the final rule on August 15, 1985. An important part of the rulemaking was the evaluation of how effective mined geologic repositories are for isolating high-level wastes from the environment for many thousands of years. EPA's assessments indicate that carefully designed repositories at good sites can keep long-term risks below those that would exist if (on a generic basis) the uranium ore used to create the wastes had not been mined to begin with. Accordingly, the Agency has promulgated environmental standards that would restrict projected releases from high-level waste disposal systems--for 10,000 years after disposal--to levels that should keep the risks to future generations less than the risks they would have been exposed to from the unmined ore if these wastes had not been created.

This technical report presents the methodology used to assess the long-term population risks from projected releases of waste from a geologic repository. It describes the models that the Agency developed specifically for this project and reviews the various assumptions made. The Agency expects that population distributions, food chains, and living habits may change dramatically over 10,000 years. Rather than attempt to predict such changes, this methodology uses very general models of environmental pathways that consider present values for the various parameters used in the models.

Because much of this methodology is new, and because these risk assessments are a key part of our rulemaking, the Agency published a draft of this report. Comments were received from the public and from EPA's Science Advisory Board High-Level Radioactive Waste Disposal Subcommittee. This Subcommittee conducted an independent technical review of the EPA risk assessments during 1983, and its suggested changes have been incorporated in the methodology which is described in this final report.

I encourage users of this report to submit any comments or suggestions they might have. Such comments should be sent to: Central Docket Section (A-130); Environmental Protection Agency; Attn: Docket No. R-82-3; Washington, D.C. 20460. For additional information, please contact Mike Smith at (205) 272-3402; Office of Radiation Programs; Eastern Environmental Radiation Facility; Environmental Protection Agency; 1890 Federal Drive, Montgomery, Alabama 36109.

Sheldon Meyers

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Office of Radiation Programs (ANR-458)

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SUMMARY

As part of its program to develop environmental standards for disposal of high-level radioactive wastes, the Environmental Protection Agency estimated population health risks for 10,000 years after disposal in mined geologic repositories (EPA85a). This report describes the mathematical models EPA ~~we~~ formulated to calculate the environmental risk commitments (ERC's--fatal cancers and serious genetic effects to all generations) that could occur as a result of releases from such repositories. The report also identifies the data EPA ~~we~~ chose to use in these models and presents our estimates of the premature fatal cancers caused per unit of radionuclide released to the accessible environment. These estimates have been used to select the containment requirements in the Agency's final disposal standards (40CFR191).

In performing these long-term assessments of population health effects, we recognize that it is pointless to try to make precise projections of the actual risks from repositories. Population distributions, food chains, living habits, and technological capabilities will undoubtedly change in major ways over 10,000 years. Unlike geological processes, they can be realistically predicted only for relatively short times. Accordingly, we formulated very general models of environmental pathways, and we assumed population sizes and characteristics similar to those of today. In particular, we usually avoided the detailed analytical techniques that would be appropriate for near-term environmental assessments of specific facilities.

The models described in this report consider risks to populations, as opposed to risks to individuals. Therefore, individual risks caused by potential releases from a repository cannot be determined from these analyses. However, the Background Information Document (BID) for 40 CFR 191 (EPA85b) describes analyses that assess individual risks from several types of releases.

S.1 The Methodology

The Agency's risk assessment reports (EPA85a, Sm82) identified four ways that radionuclides might be released to environmental pathways: to surface water (e.g., a river) through groundwater; to an ocean through surface water; to a land surface directly; or to multiple pathways after the very unlikely possibility of disruption by a volcano or a meteorite. For each of these four release modes, we modeled the ways that radionuclides can move through the geosphere and the biosphere to the population, and we estimated the intake by or exposure to the population through each of these environmental pathways. We then applied risk conversion factors per unit intake or per unit external exposure to estimate fatal cancers and serious genetic effects to all generations per curie released to the accessible environment.

Following procedures similar to those used for computing environmental dose commitments, we calculated the total health effects for the entire population exposed to the releases from a repository, rather than terminate the calculation at some arbitrary distance from the repository. A time integration was performed to sum the health effects from the time the repository is sealed ("disposal") until a specified time in the future (usually 10,000 years after disposal). The following sections summarize the procedures we used to calculate the population intake of radioactivity for the internal pathways or integrated population exposure for the external pathways for each of the four release modes. The population intakes of and exposures to radioactivity are converted to population ERC's by multiplying by the appropriate risk conversion factor.

S.1.1 Releases to a River (Surface Water)

In the surface water release model, the repository containment is breached--after some initial period--and groundwater circulates through the repository into the surrounding geologic media and eventually to an aquifer. The aquifer then flows underground until it intersects a river. To determine the total release to the river, we developed and integrated an equation describing the release rate. The integrated form of the release equation was then used to compute integrated river water concentrations for use in the following environmental pathway models:

Drinking Water. We assumed that the population of interest receives drinking water from the river with no reduction in radionuclide concentrations due to water treatment. The intake rate for surface water by an individual is combined with the ratio of the population drinking water to the river flow rate to obtain an estimate of the total intake of the radionuclide by the population per curie of the radionuclide released to the river.

Ingestion of Fish. We assume that fish caught in the river take up radioactivity from the water. By calculating the concentration of radionuclides in the fish and by multiplying by estimated fish ingestion rates by the population, we determined the total population intake, due to comsumption of fish, of the radionuclides released from the repository.

Ingestion of Food Raised on Irrigated Land. We assume that river water containing radionuclides from the repository is used to spray irrigate farm land by direct deposition onto the crops and the land surface below the crops. Furthermore, we assume that irrigated plants that have incorporated radionuclides through their leaves and root systems are consumed by humans as food--or are consumed by either dairy or beef cattle that transfer radionuclides to milk and meat. Ingestion rates of these various food products are used to estimate the total radionuclide intake by the population due to using river water for irrigation.

Inhalation of Resuspended Material. Some of the radionuclides deposited on the soil by irrigation are resuspended into the air. Using a resuspension factor and the integrated soil surface concentration, we estimated the resulting integrated concentration of radionuclides. We calculated the population intake of radionuclides using a standard inhalation rate and the size of the population.

External Exposure from Air Submersion. The radionuclides resuspended into the air can cause submersion exposures to the population. These exposures are analogous to the integrated air concentration to which the population is exposed and are calculated from the integrated air concentration, the population density, and a shielding and occupancy factor.

External Exposure from Ground Contamination. The radioactive material deposited on the ground during irrigation can also cause external exposures to persons in the area. Throughout the irrigation period, radionuclides continue to build up on the ground until either irrigation stops or equilibrium with losses through the soil is reached. The methods for estimating these exposures are similar to those applied for air submersion.

S.1.2 Releases to an Ocean

We assume that releases to a river system subsequently discharge into the ocean. Since we do not consider radionuclide decay during travel in the river or depletion of the radionuclide inventory due to river water use or sedimentation, the radionuclide releases to the ocean are identical to the releases to a river. Our model of the ocean pathway has two compartments: a shallow upper layer in which it is assumed that all edible seafood is grown and a lower layer that includes the remainder of the ocean. We developed coupled differential equations whose solutions express the quantities of radionuclides in these two compartments. The equation for the upper compartment inventory was divided by the volume of the compartment to determine the time-dependent concentation of radionuclides in the upper layer. This concentration was then used to estimate the amount of radioactivity taken up by the ocean fish and shellfish consumed by the population.

S.1.3 Releases Directly to Land Surface

For the land surface pathway models, we assume that some of the radioactive waste from the repository is brought to the surface after an event such as inadvertant intrusion while drilling for resources. Such releases to the surface are assumed to be over a small area and a short period of time--so that they can be modeled as instantaneous point sources. The mechanisms distributing the material to humans are resuspension and subsequent dispersion in the atmosphere. When the initial release to the land surface is determined, a time-dependent release rate to the air is estimated using a simple exponential model that depletes the land surface source to account for resuspension and radioactive decay. This release rate is applied in conjunction with an atmospheric dispersion equation to predict air concentrations as a function of time and distance from the source; these air concentrations are then used to estimate ground surface concentrations as a function of time and distance. Once ground surface concentrations are determined, the techniques used to calculate population intake are similar to those described for the river release mode. The pathways considered for releases to land surface are: (1) ingestion of food raised on land contaminated with radionuclides, including food crops, milk, and meat; (2) inhalation of resuspended radionuclides; (3) external exposure due to air submersion; and (4) external exposure due to ground contamination.

S.1.4 Releases Due to a Volcanic Eruption or Meteorite Impact

Releases caused by the extremely unlikely events of disruption by volcanoes or meteorites can be to the land surface and directly to the air. For the material released to the land surface, we used the methodology described for the land surface release mode. For the material released to the air, we assume that the radioactivity would be quickly dispersed in such a manner that it would eventually be distributed uniformly within the troposphere. The airborne material is divided into the fraction over land and the fraction over water--using the ratio of earth land surface and earth water surface. We then used compartment models, with their systems of coupled differential equations, to estimate the quantity of radionuclides reaching the land surface or ocean. Finally, we estimated the amount of radioactivity or radiation exposure reaching people through the same pathways described for the land surface or the ocean, respectively.

S.2 Results

The specific equations used for each of the steps in the methodology discussed above, and the parameters used in our current application of this methodology, are presented in the body of this report. Chapter 6 of the report describes the detailed results of our assessment, indicating the population health effects estimated--per curie released to the accessible environment--for each of the environmental pathways considered under each release mode (a total of 30 pathways). Chapter 7 discusses the consideration of parameter uncertainty that EPA took into account in obtaining the results discussed in Chapter 6. The following table displays the total fatal cancers per curie release values calculated for each of the four release modes. The most stringent of these sets of values--that for surface water--was used in calculating the release limits for the containment requirements of the Agency's proposed disposal standards (EPA85b).

TABLE S-1:
Fatal cancers per curie released for different release modes

Nuclide	Releases to a River	Releases to an Ocean	Releases to Land Surface	Releases due to Violent Interactions*
C - 14	5.83E-02	5.83E-02	5.83E-02	5.83E-02
NI- 59	4.61E-05	1.70E-06	6.79E-07	2.89E-05
SR- 90	2.25E-02	6.14E-06	3.76E-05	1.16E-03
ZR- 93	1.51E-04	8.59E-06	2.26E-05	1.22E-04
TC- 99	3.65E-04	3.17E-06	5.65E-08	1.99E-04
SN-126	1.05E-02	2.07E-03	1.38E-03	2.73E-02
I -129	8.07E-02	1.43E-04	3.96E-03	5.57E-02
CS-135	7.73E-03	2.61E-05	5.75E-04	4.91E-03
CS-137	1.07E-02	2.28E-05	2.19E-05	3.39E-03
SM-151	9.38E-06	4.01E-07	6.71E-08	4.72E-06
PB-210	1.18E-01	6.91E-03	1.52E-04	4.31E-02
RA-226	1.63E-01	5.40E-03	5.62E-03	7.20E-02
RA-228	2.41E-02	1.07E-04	1.57E-05	2.78E-02
AC-227	6.67E-02	1.94E-03	1.24E-04	3.82E-02
TH-229	3.49E-02	2.71E-02	1.90E-02	5.06E-02
TH-230	5.38E-01	1.87E-01	3.86E-01	1.26E+00
TH-232	3.40E-01	4.32E-02	3.76E-01	3.73E-01
PA-231	1.48E-01	1.70E-03	2.36E-02	1.28E-01
U -233	2.15E-02	1.90E-04	7.51E-04	7.75E-03
U -234	1.96E-02	1.73E-04	6.54E-04	5.94E-03
U -235	2.17E-02	1.89E-04	8.42E-04	8.27E-03
U -236	1.85E-02	1.64E-04	6.18E-04	5.62E-03
U -238	2.06E-02	1.83E-04	6.90E-04	5.67E-03
NP-237	7.95E-02	7.03E-03	1.21E-04	2.83E-02
PU-238	4.23E-02	4.06E-04	3.10E-04	2.07E-02
PU-239	4.97E-02	2.19E-03	6.23E-03	1.20E-02
PU-240	4.84E-02	1.91E-03	5.22E-03	1.15E-02
PU-241	2.17E-03	9.24E-06	2.50E-06	9.36E-04
PU-242	4.79E-02	2.18E-03	6.34E-03	1.09E-02
AM-241	5.42E-02	3.74E-03	1.05E-03	2.54E-02
AM-243	5.72E-02	1.09E-02	2.45E-03	3.40E-02
CM-245	1.01E-01	2.29E-02	8.08E-03	6.09E-02
Cm-246	4.99E-02	1.01E-02	3.54E-03	2.89E-02

*For example, interactions of a metorite or a volcanic eruption with a repository.

Chapter 1: INTRODUCTION

1.1 Background Information

This report describes the methodology used by EPA to estimate future cancer fatalities to populations due to radionuclides that escape to the environment from a high-level waste repository.* Mathematical models for estimating these cancer fatalities are presented. An estimate of the fatal cancers per curie release to the accessible environment for several radionuclides is needed for use in setting curie release limits in EPA's high-level waste standard and these estimates are given in Chapter 6.

We obtained population cancer fatality estimates in a manner consistent with methods used by others to calculate population environmental dose** commitments. The difference is that in our calculations we used risk conversion factors in place of dose conversion factors. Hence, the equations given in Chapter 3 are used to determine population "environmental risk commitments" rather than environmental dose commitments. The nomenclature used to specify environmental risk commitment is FHE_{np}*** where the subscripts n refer to radionuclide and p to pathway. However, since the concept of environmental dose commitment will be most familiar to the reader, the basis for its calculation is reviewed below.

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*Unless we state otherwise, we are estimating "excess" cancer fatalities, i.e., those caused by elevating radiation levels and in addition to those from other causes.

**For simplicity, the term "dose" will be used to denote "dose equivalent."

***The variables used in the equations in this report are defined in the nomenclature, p. N-1 ff.

The environmental dose commitment is identical to collective dose commitment, S, as used in ICRP Publication 26, "Recommendations of the International Commission on Radiological Protection" (ICRP77) and the report entitled "Sources and Effects of Ionizing Radiation" (UN77). Mathematically, the environmental dose commitment is expressed as

$$S = \int_0^\infty \int_0^\infty D' \, N(D',t) \, dD' dt \qquad (1.1\text{-}1)$$

where D' is the dose commitment rate and N(D',t) dD' is the number of people receiving a dose commitment rate between D' and D'+dD' at time t. The incomplete collective dose commitment, S(t), is defined as follows:

$$S(t) = \int_0^t \int_0^\infty D' \, N(D',t'') \, dD' dt'' \, . \qquad (1.1\text{-}2)$$

The term "dose commitment rate" as used here means the 50-year dose commitment rate to individuals due to intake in the case of internal emitters, or, in the case of external exposure, simply the annual dose rate.

In this assessment, the population of interest is the world population, which is assumed to be constant over the time period, t. Specifically, we assume that

$$\int_0^\infty N(D',t) dD' = N(t) = 10^{10} \text{ persons (UN77).} \qquad (1.1\text{-}3)$$

Other EPA reports, besides this one, discuss dosimetry and health risk analysis for the EPA high-level waste standard (EPA85a, EPA85b). In these reports, individual and population risk assessments for various scenarios which release radionuclides from a repository, are discussed. The risk assessment report (EPA85a) discusses risk assessments used to determine repository release limits for inclusion in the EPA standard.

1.2 Pathways Considered

These analyses consider four general modes of radionuclide releases from a waste repository: releases to a river, to an ocean, to a land surface, and from a volcanic or meteorite interaction with a waste repository. The releases to a river, an ocean, or a land surface can be caused by any number of events. For example, drilling a mining borehole through a repository could allow groundwater to leach radionuclides from the radioactive waste. The radionuclides could be transported to a river and subsequently to an ocean. A variation of this scenario would be radionuclide releases directly to the land surface by a drilling event. Volcanic eruption or meteorite impact have a lower probability of occurrence than those events discussed above. This class of events generally results in violent release of radioactive material directly to the land surface and to the air. These four release modes are subdivided into a total of 30 pathways, as listed in Table 1-1.

TABLE 1-1:
Release modes and environmental pathways

Release Mode	Pathways Included in this Release Mode	Pathway Number
Releases to River	Drinking Water Ingestion	1
	Freshwater Fish Ingestion	2
	Food Crops Ingestion	3
	Milk Ingestion	4
	Beef Ingestion	5
	Inhalation of Resuspended Material	6
	External Dose-Ground Contamination	7
	External Dose-Air Submersion	8
Releases to Ocean	Ocean Fish Ingestion	9
	Ocean Shellfish Ingestion	10
Releases Directly to Land Surface	Food Crops Ingestion	13
	Milk Ingestion	14
	Beef Ingestion	15
	Inhalation of Resuspended Material	12
	External Dose-Ground Contamination	16
	External Dose-Air Submersion	11
Releases Due to Volcano/ Meteorite Interaction		
Releases Directly to Land	Food Crops Ingestion	25
	Milk Ingestion	26
	Beef Ingestion	27
	Inhalation of Resuspended Material	24
	External Dose-Ground Contamination	28
	External Dose-Air Submersion	23
Releases to Air Over Land	Food Crops Ingestion	19
	Milk Ingestion	20
	Beef Ingestion	21
	Inhalation of Dispersed and Resuspended Material	17
	External Dose-Ground Contamination	22
	External Dose-Air Submersion	18
Releases to Air Over Ocean	Ocean Fish Ingestion	29
	Ocean Shellfish Ingestion	30

1.3 Approach to Calculations

The goal of the analysis for each specific pathway is to estimate the population cancer fatalities committed to time t per unit nuclide release to the accessible environment*. After the normalized values are determined as a function of nuclide and pathway, a summation over pathways may be performed. The result is a normalized set of ERCs, for a release mode, which are a function only of nuclide. With this approach, these normalized ERCs can be used to predict the total ERC for various scenarios which lead to releases to the biosphere from a repository, without a "reanalysis" for each new event.

Developing pathway computation models involves applying the mechanisms for leakage of radionuclides from a repository and predicting the processes that cause these radionuclides to disperse in the environment. These models and methods are discussed in more detail in Chapters 2 and 3. In general, the pathway equations integrate the risks over all persons exposed from the time the material is placed in the repository until time, t, in the future, to yield the incomplete environmental risk commitment. Conceptually, the incomplete environmental risk commitment, ERC(t), can be defined in a manner analogous to equation 1.1-2, i.e.,

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*According to our terminology in section 1.1, these population cancer fatalities are the normalized, incomplete environmental risk commitments. For ease of reference, the acronym used throughout the report to refer to these quantities will be ERC.

$$ERC(t) = \int_0^t \int_0^\infty R' \, N(R',t) \, dR' dt \, . \qquad (1.3\text{-}1)$$

where R' is the risk commitment rate and N(R',t)dR' is the number of people receiving a risk commmitment rate between R' and R'+dR' at time t.

Chapter 2: SOURCE TERMS

We use simple transport models to estimate the movement of radionuclides from the repository into the accessible environment. Different scenarios are applied in developing the source term models for the four release modes. For some of the release modes, several different mathematical models for determining releases to the environment are possible. In this report, only one release model has been addressed for each release mode. The environmental pathway calculations for other release models would be treated in a manner similar to the methods discussed in this report. A more complete discussion of source terms is included in the risk assessment report (EPA85a).

2.1 River Source Terms

For the river pathway, we assume the radionuclides remain in the repository for the initial delay period, t_{er}, following placement.* After this time, we assume that the repository is breached and that radionuclides can be removed from the repository and eventually be transported to a river. There are several different mechanisms that determine the rate of removal of radionuclides from a repository, such as their susceptibility to leaching, water flow through the repository, solubility of the chemical compound containing the radionuclides, waste heat from the buried waste, and type of event causing the release. The methods for obtaining the specific releases for a release scenario are discussed in the risk assessment report (EPA85a). In this report, we use a radionuclide removal model limited by the rate of leaching to illustrate typical river environmental transport equations. Several other release models are plausible, but this "leaching-limited" model has been chosen because it is reasonably simple and leads to closed-form environmental pathway equations.

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*The variables used in this report are defined in the nomenclature, p. N-1 ff.

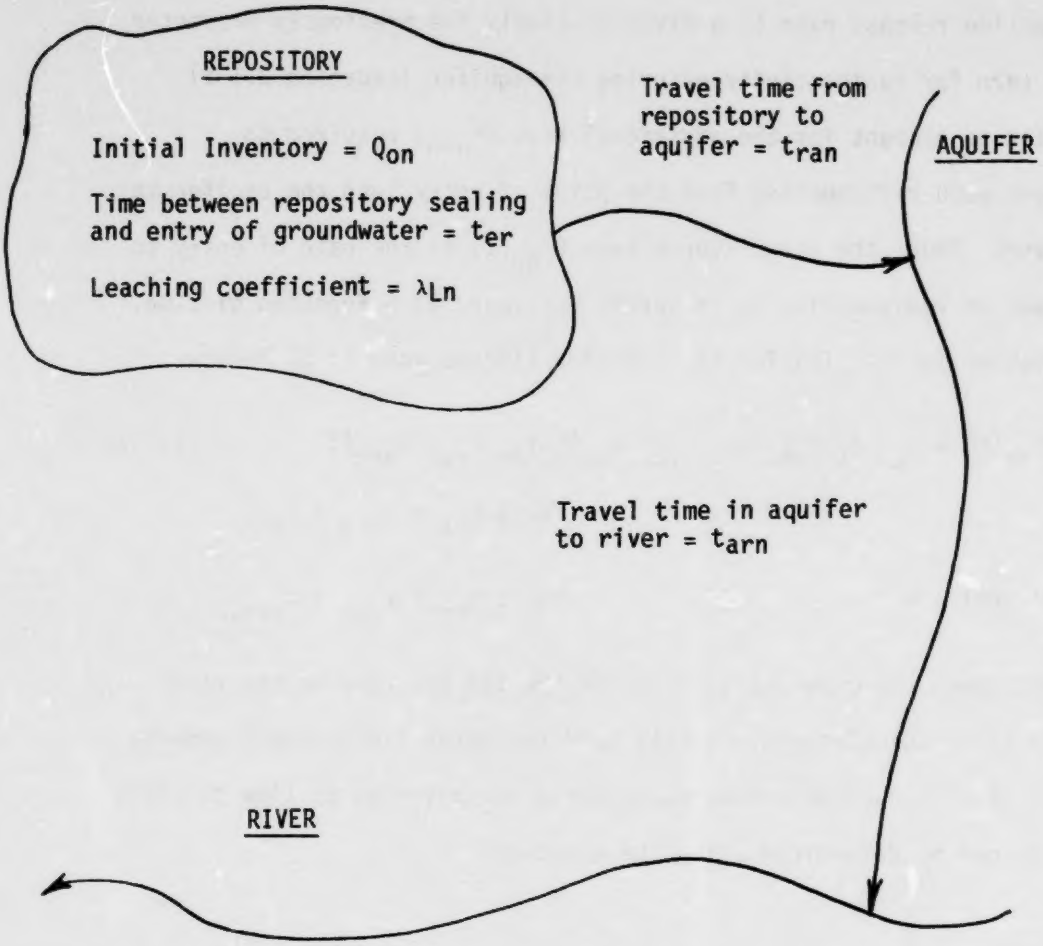
The total human intake for each river pathway is taken to be proportional to the total quantity (i.e., the integrated concentration) of the radionuclide in the river. Therefore, the time-dependent rate of entry of radionuclides to the river must be developed so that it can be integrated. We assume that the repository is breached and groundwater circulates through the repository and leaches radionuclides into the surrounding area and eventually to an aquifer after passage of additional time, t_{ran}. The radionuclide-specific source term equation that describes the time-dependent rate of entry of each radionuclide (n) into the aquifer, $Q'_{anp}(t)$, is expressed as (see Fig. 2-1)

$$Q'_{anp}(t) = \lambda_{Ln} f_L Q_{on} \exp [-\lambda_{Dn} t - \lambda_{Ln} (t-t_{er}-t_{ran})] \qquad \text{for } t>t_{er} + t_{ran}$$

and (2.1-1)

$$Q'_{anp}(t) = 0 \qquad \text{for } t \leq t_{er} + t_{ran}.$$

The aquifer flows underground for a representative distance until it intersects a river system. Radionuclides entering the aquifer are assumed to reach the river after a delay time, t_{arn}. The equation for


REPOSITORY
Initial Inventory = Q_{on}
Time between repository sealing and entry of groundwater = t_{er}
Leaching coefficient = λ_{Ln}
Travel time from repository to aquifer = t_{ran}
AQUIFER
Travel time in aquifer to river = t_{arn}
RIVER

(Note: $t_{Rn} = t_{er} + t_{ran} + t_{arn}$ = time between sealing of repository and entry of radionuclides into the river.)

Fig. 2-1. Schematic diagram for transport of radionuclides from repository to river

radionuclide release rate to a river is simply the previously presented source term for radioactivity entering the aquifer (equation 2.1-1) corrected to account for the additional time (t_{arn}) required to transport each radionuclide from the point of entry into the aquifer to the river. Thus, the river source term $Q'_{np}(t)$ is the rate of entry to the river of radionuclide n, in curies per year, as a function of time. The equation for $Q'_{np}(t)$ for the leaching-limited case is as follows:

$$Q'_{np}(t) = \lambda_{Ln}\, f_L\, Q_{on}\, \exp[-\lambda_{Dn}t-\lambda_{Ln}(t-t_{er}-t_{ran}-t_{arn})] \qquad \text{for } t>t_{er}+t_{ran}+t_{arn} \qquad (2.1\text{-}2)$$

and

$$Q'_{np}(t) = 0 \qquad \text{for } t\leq t_{er}+t_{ran}+t_{arn}.$$

Fig. 2-2 shows the exponential shape of the release rate to the river.

In later calculations, we will need the total (integrated) amount, $Q_{np}(t)$, of radionuclide n that has entered the river up to time t. This quantity can be determined using the equation

$$Q_{np}(t) = \int_0^t Q'_{np}(t'')\, dt'' . \qquad (2.1\text{-}3)$$

Substituting equation 2.1-2 into equation 2.1-3 and integrating, we obtain

$$Q_{np}(t) = \frac{f_L \lambda_{Ln} Q_{on}}{\lambda_{Dn}+\lambda_{Ln}} \begin{bmatrix} \exp[-\lambda_{Dn}t_{Rn}] \\ -\exp[\lambda_{Ln}t_{Rn} -(\lambda_{Dn}+\lambda_{Ln})t] \end{bmatrix} \qquad \text{for } t>t_{Rn} \qquad (2.1\text{-}4)$$

and

$$(t_{Rn} = t_{er} + t_{ran} + t_{arn})$$

$$Q_{np}(t) = 0 \qquad \text{for } t\leq t_{Rn} .$$

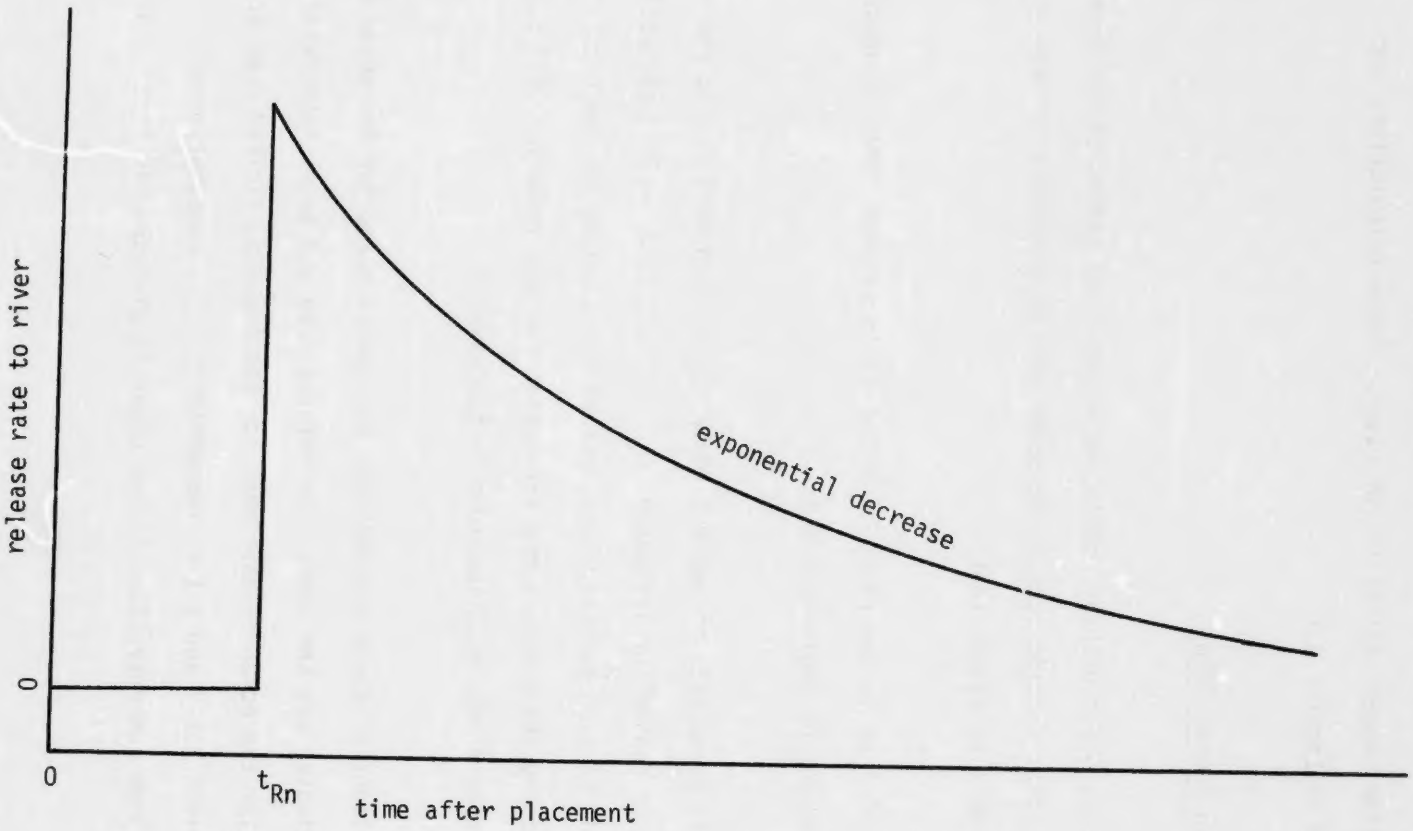


Figure 2-2. Release rate to river as a function of time after placement of radioactive waste in repository

Once the source term to the river is determined, we can calculate radionuclide concentrations in the river. These calculations are discussed in Section 3.1.

2.2 Ocean Source Terms

For the ocean pathway models, we assume that radionuclides from a waste disposal facility reach the ocean only by transport through a river system. We also assume that

-- travel time in the river to the ocean is so small that radionuclide decay may be neglected; and

-- it is acceptable to neglect depletion of radionuclides in the river due to removal by irrigation and sedimentation. (This assumption leads to a conservative estimate of the quantity of radionuclides reaching the ocean, since irrigation and sedimentation will remove a portion of the radionuclides in the river.)

In light of these assumptions, the source terms for the ocean and river releases are the same. The release rate and total integrated release for the ocean release mode for the leaching-limited case are given by equations 2.1-2 and 2.1-4, respectively. The computation of radionuclide concentrations in the ocean is discussed in Section 3.2.

2.3 Land Surface Source Terms

For the river and ocean release mode, the source term model employed predicts a continuous long-term release of radionuclides to the biosphere after leakage begins at a waste repository. The events hypothesized for the land surface release mode are those which could cause radionuclides from the repository to be brought directly to the earth's surface in a short time period -- such as drilling a well into or through a waste repository. We assume that these events result in an instantaneous release of some fraction, f_{LS}, of the contents of the repository to the land surface at a time, t_L, after emplacement of waste in a repository. The release is assumed to be to a small area of land, which can be considered as a point source for calculation of the resuspension from ground to air and the subsequent redistribution in the environs. The ERC pathway analyses for the land surface release mode are discussed in Section 3.3.

2.4 Volcano/Meteorite Interaction Source Terms

We assume that a violent volcanic eruption or a meteorite impact liberates materials from the repository in a short time. A fraction, f_{LL}, of this material goes directly to the land surface and the rest is released directly to the air. That released to the land surface is conservatively assumed to be distributed in a small area, and the methodology for treating the redistribution of this material in the environment is the same as that used for the land surface release. We assume that the material released to the air is dispersed in such a manner that it is, eventually, distributed uniformly in the troposphere. The airborne material is divided into the fraction over land, f_{AL}, and the fraction over water, f_{AW}, using the ratio of earth land surface area to total earth area and earth water surface area to total earth area.

The methodology used to predict distribution of radionuclides in the environment and to analyze the ERC pathways for the volcano/meteorite release mode is discussed in Section 3.4.

Chapter 3: ENVIRONMENTAL TRANSPORT AND RISK MODELS

This section contains the specific mathematical models used to calculate environmental risk commitment (ERC) for each pathway and a discussion of the rationale for each model.* The sources of the data used in applying the models are discussed in Chapters 4 and 5.

The models used in these analyses are simple, but we believe they are appropriate for setting generally applicable environmental standards. There is a large amount of uncertainty in some of the parameters (see Chapter 7 for a discussion of uncertainty). For this reason, it would not be appropriate to apply more sophisticated models. When possible, the end results of existing, more detailed model evaluations are used.

We have used to the greatest extent possible what we consider to be realistic assumptions in developing these environmental pathway models. When we use conservative assumptions, we note them in the discussion of the affected pathway models. In this regard, one potentially conservative assumption affects all the environmental pathway models: the computation of the environmental risk commitment using the linear, non-threshold assumptions to estimate risks to the exposed population.** The procedure for performing these calculations involves computation of risks to the entire exposed population, some of whom will experience very low dose equivalent rates. To the extent that there may be a threshold below which the dose equivalent does not result in a fatal cancer risk, these calculations are conservative. However, application of the linear, non-threshold theory is a prudent assumption since the existence of a threshold has not been proved. It is applied by essentially all regulatory agencies in the radiation protection field.

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*The variables used in the equations in this report are defined in the nomenclature, p. N-1 ff.

**The environmental risk commitment and the linear, non-threshold theory are discussed more fully in Chapters 1 and 4 and Appendices F and G.

Terms used in the equations in the following sections are defined in the "Nomenclature", except where a term is used only locally in a section. In those instances, the terms may be defined in the sections where they are used.

In the following sections, the equations that are discussed are for the normalized population environmental risk commitment (ERC) per unit of radionuclide release to the accessible environment for the 30 pathways, i.e., for all pathways, we calculate the possible fatal cancers per unit of radioactivity release (or the "normalized" fatal cancers). This is done by multiplying normalized radionuclide intake by the appropriate internal fatal cancer risk factor for the internal pathways (inhalation or ingestion) and by multiplying normalized radionuclide exposure by the appropriate external fatal cancer risk factor for the external pathways (exposure from ground surface irradiation or from air submersion). In this report, the internal and external fatal cancer risk factors are denoted as FCF_{np} (fatal cancer factor for radionuclide n and pathway p).

The cancer fatalities for the various pathways within a release mode may be summed to obtain total fatal cancers per unit release to the accessible environment for each specific radionuclide. The mathematical formulation for this process can be written as

$$\frac{FHE_n}{Q_n} = \sum_p \frac{RIE_{np}}{Q_{np}} (FCF_{np}) \tag{3.1-1}$$

where the summation over pathways (p) extends over the number of pathways included in a release mode.

3.1 Releases to a River

3.1.1 General Considerations

The methods for determining the release of radionuclides from the repository to the river are discussed in Section 2.1. Using these releases, the radionuclide concentrations in the river are computed by dividing the average yearly radionuclide source terms by the average river flow rate. Radionuclides from the river expose the local population through drinking water, ingestion of freshwater fish, irrigation pathways (including ingestion of food crops, milk, and beef), inhalation of resuspended radioactivity, and direct radiation dose from ground surfaces and from air. These specific pathways are discussed below.

In estimating the ERC for these pathways, we did not consider removal of radionuclides from the river water by sedimentation. There is no question that certain radionuclides deposit into sediments in slowly flowing rivers and lakes, and, during the short-term, sedimentation lowers river water concentrations. However, over the long time period considered in these calculations, massive floods (100 and 1000 year floods, etc.), may occur so that much of the deposited activity may be resuspended from the sediment and distributed on farm land. For this reason and because of the difficulty involved in modeling sedimentation and subsequent resuspension during floods, we chose what we consider to be a conservative position and did not model radionuclide loss from river water due to sedimentation. Also, we conservatively assumed that the entire river is contaminated so that all persons in the vicinity of the river are subjected to the radiation risk.

3.1.2 Drinking Water Ingestion [pathway number (p) = 1]

The drinking water supply for a population can be taken from rivers, lakes, or groundwater sources. While selected aquifers may be contaminated by leaching of radionuclides from a high-level waste repository, we believe that repositories will be sited far enough away from potable groundwater supplies to preclude the contamination of potable groundwater for significant populations. Thus we have assumed that contaminated drinking water would come only from rivers or lakes. For the long periods chosen for this analysis, lake and river water concentrations should become equal. Thus, we have restricted our analysis to consideration of consumption of river water by the population.

The annual risk commitment to an individual is given by the product of the amount of river water drunk ($I_w . f_{sw}$), the radionuclide concentration in drinking water ($Q'_{np} . f_{wt}/R$), and the risk conversion factor (FCF_{np}) in the equation:

$$RIV'_{np} = \frac{Q'_{np} \ I_w \ FCF_{np} \ f_{sw} \ f_{wt}}{R} . \qquad (3.1.2\text{-}1)$$

The annual population risk commitment can be expressed as the annual individual risk commitment multiplied by the number of persons drinking the water, or

$$FHE'_{np} = RIV'_{np} \ P_R = \frac{Q'_{np} \ I_w \ FCF_{np} \ f_{sw} \ f_{wt} \ P_R}{R} ^{*} . \qquad (3.1.2\text{-}2)$$

By integrating the above expression over time we obtain

$$FHE_{np} = \frac{Q_{np} \ I_w \ FCF_{np} \ f_{sw} \ f_{wt} \ P_R}{R} \qquad (3.1.2\text{-}3)$$

where

$$\int_0^t FHE'_{np}(t'') \ dt'' = FHE_{np} \qquad (3.1.2\text{-}4)$$

and

$$\int_0^t Q'_{np}(t'') \ dt'' = Q_{np} . \qquad (3.1.2\text{-}5)$$

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*In this equation, the only time dependent term is Q'_{np}.

Upon dividing both sides of equation 3.1.2-3 by the integrated release to the river, Q_{np}, we obtain

$$\frac{FHE_{np}}{Q_{np}} = \frac{P_R \ I_W \ FCF_{np} \ f_{sw} \ f_{wt}}{R} \ . \tag{3.1.2-6}$$

After obtaining values for the parameters, equation 3.1.2-6 can be used to calculate the normalized ERC to the population from the ingestion of river water. Parameter values are discussed in Chapters 4 and 5. While provisions were included in the model to allow for removal of nuclides by water treatment plants, the modifying factor, f_{wt}, was not used in these analyses. This is a conservative assumption.

3.1.3 Freshwater Fish Ingestion (p=2)

The annual risk commitment to an individual is given by the amount of fish eaten (I_f), the radionuclide concentration in the river water (Q'_{np}/R), a concentration factor expressing the concentration of radionuclides in fish compared to the concentration in water (CF_{np}), and the risk conversion factor (FCF_{np}):

$$RIV'_{np} = \frac{Q'_{np} \ CF_{np} \ I_f \ FCF_{np}}{R} \ . \tag{3.1.3-1}$$

For some radionuclides, the fish concentration factors may be based on total radioactivity in the fish rather than only radioactivity in the fish muscle (the tissues used for food). In cases where this occurs and where the radionuclide tends to concentrate in the parts of the fish which are not eaten, the CF factors will be conservatively high.

The annual population risk commitment can be expressed as the annual individual risk commitment multiplied by the number of persons eating fish:

$$FHE'_{np} = RIV'_{np} \; P_{FF} = \frac{Q'_{np} \; CF_{np} \; I_f \; FCF_{np} \; P_{FF}}{R} \; . \qquad (3.1.3\text{-}2)$$

By integrating this expression over time, using the same procedures as for the drinking water pathway, we obtain

$$FHE_{np} = \frac{Q_{np} \; CF_{np} \; I_f \; FCF_{np} \; P_{FF}}{R} \; , \qquad (3.1.3\text{-}3)$$

where the method for obtaining FHE_{np} and Q_{np} is given in equations 3.1.2-4 and 3.1.2-5. Upon dividing both sides of equation 3.1.3-3 by the integrated release to the river, Q_{np}, we obtain

$$\frac{FHE_{np}}{Q_{np}} = \frac{CF_{np} \; P_{FF} \; I_f \; FCF_{np}}{R} \; . \qquad (3.1.3\text{-}4)$$

This equation is for freshwater fish from rivers, since we assume that all freshwater fish eaten by the population come from rivers. Similar to the drinking water pathway, this assumption allows us to ignore additional pathways for fish consumption associated with lakes. Using the data discussed in Chapters 4 and 5, equation 3.1.3-4 can be used to calculate the ERC to the population from ingestion of freshwater fish.

3.1.4 Food Ingestion

Food crops ($p=3$)
Milk ($p=4$)
Beef ($p=5$)

The annual risk commitment to an individual from consuming foods raised on irrigated land is a product of the concentration of radionuclides in the river water (Q'_{np}/R), the irrigation rate (W), a conversion factor to express the radionuclide intake by an individual per unit deposition to the ground surface (RI_{np}), and the risk conversion factor (FCF_{np}):

$$RIV'_{np} = \frac{Q'_{np} \; W \; RI_{np} \; FCF_{np}}{R} \; . \tag{3.1.4-1}$$

The annual population risk commitment can be expressed as the annual individual risk commitment multiplied by the number of persons being fed a particular food crop raised on irrigated land. The size of the population eating irrigated food crops can be determined by multiplying the number of persons who can be fed by raising the food crop on a unit area of land (CP_p), the area of the irrigated land (A), and a weighting factor that expresses the fraction of irrigated land used for a particular crop (f_p):

$$P_{Fp} = CP_p \; A \; f_p \; . \tag{3.1.4-2}$$

Then the annual population risk commitment is

$$FHE'_{np} = RIV'_{np} \; P_{Fp} = \frac{Q'_{np} \; W \; RI_{np} \; FCF_{np} \; CP_p \; A \; f_p}{R} \; . \tag{3.1.4-3}$$

By integrating this expression over time we obtain

$$FHE_{np} = \frac{Q_{np} \; W \; RI_{np} \; FCF_{np} \; CP_p \; A \; f_p}{R} \; . \tag{3.1.4-4}$$

The ratio (W/R) is needed for each food pathway. We can write the relationship

$$WA = f_R R \qquad (3.1.4\text{-}5)$$

where A is the area of land irrigated (m^2).

Rearranging, we have

$$\frac{W}{R} = \frac{f_R}{A} \;, \qquad (3.1.4\text{-}6)$$

which can be substituted into equation 3.1.4-4.

As mentioned previously, RI_{np} is the intake of radionuclide n by standard man for crop p for an acute deposition to the surface (Ci intake per Ci/m^2 deposited on the soil surface). Values for RI_{np} are listed in Chapter 5 (Table 5-3) and the methods and data used to determine values for RI_{np} for food products are discussed in detail in Appendix C.

The mathematical models used to determine values for RI_{np} are similar to those used by the U.S. Nuclear Regulatory Commission (NRC77) and those used by EPA in the AIRDOS-EPA computer program (Mo79). One significant change was made to the EPA models, which is included in determining the values for RI_{np}. The loss of radionuclides from the soil root zone was taken into account in computing uptake of radionuclides into plants through plant root systems*. This loss mechanism from the soil can be important for long-lived radionuclides.

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*This change was made to the AIRDOS-EPA computer code after the program manual (Mo79) was prepared. The loss mechanism is not described in the program manual.

The values of RI_{np} include a component for direct deposition to plant foliage and a component for uptake from the soil via the plant root systems. Therefore, the results are representative of spray irrigation. Risks associated with ditch irrigation will be conservatively high since direct deposition to crop surfaces is included. The only transfer mechanism for ditch irrigation is uptake by the crops from the soil.

Substituting (f_R/A) for (W/R) in equation 3.1.4-4, the irrigation area, A, cancels out and we obtain the following expression for the ERC to the local population due to the ingestion of crops:

$$\frac{FHE_{np}}{Q_{np}} = f_R \ f_p \ RI_{np} \ CP_p \ FCF_{np} \ . \qquad (3.1.4\text{-}7)$$

We make the conservative assumption that all irrigated land is used to produce edible food crops; in fact, some would probably be used to raise non-food crops. Conversely, there will be some food crops whose roots extend below the assumed 15 cm soil root zone such that they can take up radioactivity from the soil below this depth. For these cases, our use of a λ_{Sn} based on a 15 cm soil root zone is not conservative.

3.1.5 Inhalation of Resuspended Material (p=6)

The annual risk commitment to an individual from inhalation occurs due to material being deposited on the ground surface by irrigation and subsequently resuspending from the ground surface into the air. This annual risk commitment is the product of the air concentration (X_{Rn}), the inhalation rate of an individual (I_B), and the risk conversion factor (FCF_{np}):

$$RIV'_{np} = X_{Rn}\ I_B\ FCF_{np}\ . \qquad (3.1.5\text{-}1)$$

The annual population risk commitment is the annual individual risk commitment multiplied by the number of persons inhaling air containing radionuclides:

$$FHE'_{np} = RIV'_{np}\ P_{Ap}\ . \qquad (3.1.5\text{-}2)$$

The number of persons subject to inhalation can be determined by multiplying the population density for the irrigation area by the size of the area:

$$P_{An} = PD_p\ A\ . \qquad (3.1.5\text{-}3)$$

Since the exposed population was confined to persons within the irrigation area, the implicit assumption for equation 3.1.5-3 is that the radioactive material deposited during irrigation remains on the ground or in the air above the irrigated ground. Although, in practice, radioactive material resuspended into air would be diluted and distributed over a wider area, our approach should yield approximately the same numerical population risk commitment as the more exact and more complicated method of accounting for dispersion of radionuclides in the air beyond the irrigation area. Combining equations 3.1.5-1, 3.1.5-2 and 3.1.5-3 yields

$$FHE'_{np} = X_{Rn}\ I_B\ FCF_{np}\ PD_p\ A\ . \qquad (3.1.5\text{-}4)$$

The air concentration, $X_{Rn}(t)$, at the center of a uniformly contaminated area having a surface concentration, $\phi_n(t)$, due to resuspension of radionuclides from the ground surface is shown by Nelson (Ne78) to be

$$X_{Rn}(t) = RF\ \ \phi_n(t) \qquad (3.1.5\text{-}5)$$

where

$$RF = X_{Rn}(t)/\phi_n(t) = \lambda_R/v_{gn}\ . \qquad (3.1.5\text{-}6)$$

RF is the ratio of air concentration to soil surface concentration.

Equation 3.1.5-6 holds if the radius of the ground surface source term is large compared to the depletion distance for material resuspended to the air (i.e., this approximation is only valid for large, uniformly contaminated areas). We assume that redistribution of materials by resuspension in the contaminated area is inconsequential, which is equivalent to saying that the resuspended air concentration at a point in the contaminated area only depends on the soil surface concentration at the point of interest.

Substituting equation 3.1.5-5 into equation 3.1.5-4 yields

$$FHE'_{np} = RF\ \phi_n(t)\ I_B\ FCF_{np}\ PD_p\ A\ . \qquad (3.1.5\text{-}7)$$

By integrating equation 3.1.5-7 over time, we obtain the following expression for the inhalation pathway environmental dose commitment:

$$FHE_{np} = RF \left[\int_0^t \phi_n(t'')\, dt'' \right] I_B \, FCF_{np} \, PD_p \, A \, . \qquad (3.1.5\text{-}8)$$

Our procedures for developing the algorithm to describe soil surface concentration as a function of time must be explained. Since these high-level waste computations are for long time periods and for irrigated soil, which would be frequently tilled, we have assumed that radionuclides initially deposited on the ground surface are rapidly mixed into the plant root zone, which is assumed to be 15 cm deep. Bennett (Be76) has suggested that radioactive material resuspended into air generally comes from the top 1 cm of soil so that soil surface concentrations (the material subject to resuspension) should represent the inventory of radioactive material within the top 1 cm of soil. We have developed an algorithm to predict the inventory of radionuclides within the 15 cm soil root zone per unit surface area of soil, $\phi_{In}(t)$,* and have assumed that one-fifteenth of this inventory represents the radioactive material subject to resuspension (the ground surface concentration), i.e., $\phi_n(t) = \phi_{In}(t)/15$. The soil root zone inventory per unit surface area of soil, as a function of time, $\phi_{In}(t)$, is calculated by solving the differential equation

$$\phi'_{In}(t) = -(\lambda_{Dn} + \lambda_{Sn})\, \phi_{In}(t) + \frac{W}{R} Q'_{np}(t) \, , \qquad (3.1.5\text{-}9)$$

---

* For simplicity, $\phi_{In}(t)$ will be referred to as the root zone inventory.

where $\phi'_{In}(t)$ is the rate of change with time of the soil root zone inventory for radionuclide n; λ_{sn} is the removal constant from the soil root zone to the soil sink; and the other terms have been previously defined.

The term $-(\lambda_{Dn} + \lambda_{Sn})\,\phi_{In}(t)$ represents the rate of removal of radionuclide n from the root zone, and the term $(W/R)\,Q'_{np}(t)$ represents the rate of deposition of radionuclide n to the root zone by irrigation.

The basic assumption in the expression $\phi'_{In}(t)$ is that removal of radionuclides from the ground surface by resuspension, $\lambda_R\,\phi_{In}(t)/15$, is offset by deposition to the ground surface of the resuspended material, $X_{Rn}(t)\,v_{gn}$. Thus these terms do not appear in equation 3.1.5-9. This assumption should be adequate for a large distributed source. Substituting f_R/A for W/R as before, we can solve the differential equation 3.1.5-9 to obtain the expression for the soil root zone inventory, which is

$$\phi_{In}(t) = \frac{\lambda_{Ln} f_L Q_{on} f_R}{A(\lambda_{Ln}-\lambda_{Sn})} \begin{bmatrix} \exp\left[\lambda_{Sn} t_{Rn} - (\lambda_{Dn}+\lambda_{Sn})t\right] \\ -\exp\left[\lambda_{Ln} t_{Rn} - (\lambda_{Dn}+\lambda_{Ln})t\right] \end{bmatrix} \quad \text{for } t > t_{Rn}$$

and (3.1.5-10)

$$\phi_{In}(t) = 0 \quad \text{for } t \leq t_{Rn}$$

where, again: $t_{Rn} = t_{er} + t_{ran} + t_{arn}$.

As discussed in Section 2.1, this equation is based on a leach-rate limited release model, which is only one of several release models considered by EPA. The method used to address resuspension is based on

Nelson's (Ne78) assumptions; however, resuspension is addressed in a direct manner rather than using Nelson's method. The results of both methods should be identical. Substituting equation 3.1.5-10 into equation 3.1.5-8 (recognizing that $\phi_n(t) = \phi_{In}(t)/15$) and integrating, the resulting equation can be used to calculate the ERC to the local population due to inhalation of resuspended material from the ground surface. If one factors out the integrated source term to the accessible environment, Q_{np}, expressed in equation 2.1-4, the resulting equation for ERC per unit release to the river for inhalation of resuspended material becomes

$$\frac{FHE_{np}}{Q_{np}} = \frac{RF\ PD_p I_B\ FCF_{np}\ f_R}{15\ (\lambda_{Ln} - \lambda_{Sn})} \left[\frac{(\lambda_{Dn} + \lambda_{Ln}) \left[e^{-\lambda_{Dn} t_{Rn}} - e^{[\lambda_{Sn} t_{Rn} - (\lambda_{Dn} + \lambda_{Sn}) t]} \right]}{(\lambda_{Dn} + \lambda_{Sn}) \left[e^{-\lambda_{Dn} t_{Rn}} - e^{[\lambda_{Ln} t_{Rn} - (\lambda_{Dn} + \lambda_{Ln}) t]} \right]} - 1 \right]$$

for $t > t_{Rn}$ (3.1.5-11)

and

$$\frac{FHE_{np}}{Q_{np}} = 0$$

for $t \leq t_{Rn}$.

Note that the irrigation area cancels out of equation 3.1.5-11.

3.1.6 External Risk Commitment - Ground Contamination (p=7)

The derivation of an expression for environmental risk commitment from external radiation for material deposited on the ground is similar to the derivation for the inhalation pathway. As with the inhalation pathway, it is assumed that the deposited radionuclides are rapidly mixed into the 15 cm root zone. The equation is

$$FHE_{np} = [PD_p \cdot A]\int_0^t \phi_{In}(t")dt" \cdot [FCF_{np} \cdot GC_{np} \cdot SOF] \qquad (3.1.6\text{-}1)$$

where FCF_{np} is the external risk conversion factor for ground surface contamination in fatal cancers committed per Ci-y/m^2 integrated soil surface concentration and SOF is a shielding factor that accounts for the reduction in external dose due to household shielding and occupancy. Since the external risk conversion factors are derived assuming that all radioactive material is deposited on the soil surface, the correction factor (GC_{np}) is included in the algorithm to account for reduction in the external risk commitment due to soil shielding. The method of derivation of this correction factor is discussed more fully in Appendix C (Section C.5). The other terms have been previously defined or appear in the "Nomenclature" section. Using equation 3.1.5-10, the integrated root zone inventory is given by

$$\int_0^t \phi_{In}(t")dt" = \frac{\lambda_{Ln} f_L Q_{on} f_R}{(\lambda_{Ln}-\lambda_{Sn})A} \left\{ \frac{\exp[-\lambda_{Dn}t_{Rn}] - \exp[\lambda_{Sn}t_{Rn}-(\lambda_{Dn}+\lambda_{Sn})t]}{\lambda_{Dn} + \lambda_{Sn}} + \frac{\exp[\lambda_{Ln}t_{Rn} - (\lambda_{Dn}+\lambda_{Ln})t] - \exp[-\lambda_{Dn}t_{Rn}]}{\lambda_{Dn} + \lambda_{Ln}} \right\}$$

$$\text{for } t > t_{Rn} . \qquad (3.1.6\text{-}2)$$

Substituting the above equation into equation 3.1.6-1, and normalizing by the integrated source term to the accessible environment, using equation 2.1-4, one obtains the following expression for the ERC to a local population due to direct exposure from radionuclide n deposited in the soil:

$$\frac{FHE_{np}}{Q_{np}} = \frac{f_R \; PD_p \; FCF_{np} \; SOF \; GC_{np}}{(\lambda_{Ln} - \lambda_{Sn})} \left[\frac{(\lambda_{Dn}+\lambda_{Ln}) \left[e^{-\lambda_{Dn} t_{Rn}} - e^{[\lambda_{Sn} t_{Rn} - (\lambda_{Dn}+\lambda_{Sn})t]} \right]}{(\lambda_{Dn}+\lambda_{Sn}) \left[e^{-\lambda_{Dn} t_{Rn}} - e^{[\lambda_{Ln} t_{Rn} - (\lambda_{Dn}+\lambda_{Ln})t]} \right]} - 1 \right]$$

$$\text{for } t > t_{Rn}$$

and

$$\frac{FHE_{np}}{Q_{np}} = 0 \qquad \text{for } t \leq t_{Rn} \,. \qquad (3.1.6\text{-}3)$$

3.1.7 External Risk Commitment - Air Submersion (p=8)

The procedure for developing an equation to predict ERC from external exposure due to air submersion follows a rationale very similar to that for the ERC for external exposure due to ground contamination. The variation from the procedure described in Section 3.1.6 is that the material subject to resuspension is in the top 1 cm of soil, a resuspension factor (RF) is added to equation 3.1.6-1 to predict integrated air concentration due to resuspension and the risk factor has units of fatal cancers committed per Ci-y/m^3 integrated air concentration , i.e.:

$$FHE_{np} = \frac{[RF \; PD_p \; A]}{15} \int_0^t \phi_{In}(t'')dt'' \; [FCF_{np} \cdot SOF] \,. \qquad (3.1.7\text{-}1)$$

After substituting equation 3.1.6-2 for the integral in equation 3.1.7-1 and normalizing by the integrated source term, one obtains the following expression for the ERC to a local population due to direct exposure from radionuclide n in the air:

$$\frac{FHE_{np}}{Q_{np}} = \frac{RF\ f_R PD_p\ FCF_{np}\ SOF}{15\ (\lambda_{Ln} - \lambda_{Sn})} \left[\frac{(\lambda_{Dn}+\lambda_{Ln}) \left[e^{-\lambda_{Dn} t_{Rn}} - e^{[\lambda_{Sn} t_{Rn} - (\lambda_{Dn}+\lambda_{Sn}) t]} \right]}{(\lambda_{Dn}+\lambda_{Sn}) \left[e^{-\lambda_{Dn} t_{Rn}} - e^{[\lambda_{Ln} t_{Rn} - (\lambda_{Dn}+\lambda_{Ln}) t]} \right]} - 1 \right]$$

$$\text{for } t > t_{Rn} \qquad (3.1.7\text{-}2)$$

and

$$\frac{FHE_{np}}{Q_{np}} = 0 \qquad \text{for } t \leq t_{Rn}.$$

3.2 Releases to an Ocean*

3.2.1 General Considerations

This section describes the calculation of partial environmental risk commitments as the result of exposure to high-level waste radionuclides in the ocean, specifically for the case of a leaching-rate-limited source term where radionuclides from a geological repository reach the ocean through rivers. The model employed for these ocean pathway calculations is illustrated in Fig. 3-1. The model includes two compartments.

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*The variables used in the equations in this section are defined in the "Nomenclature" section, p. N-1 ff.

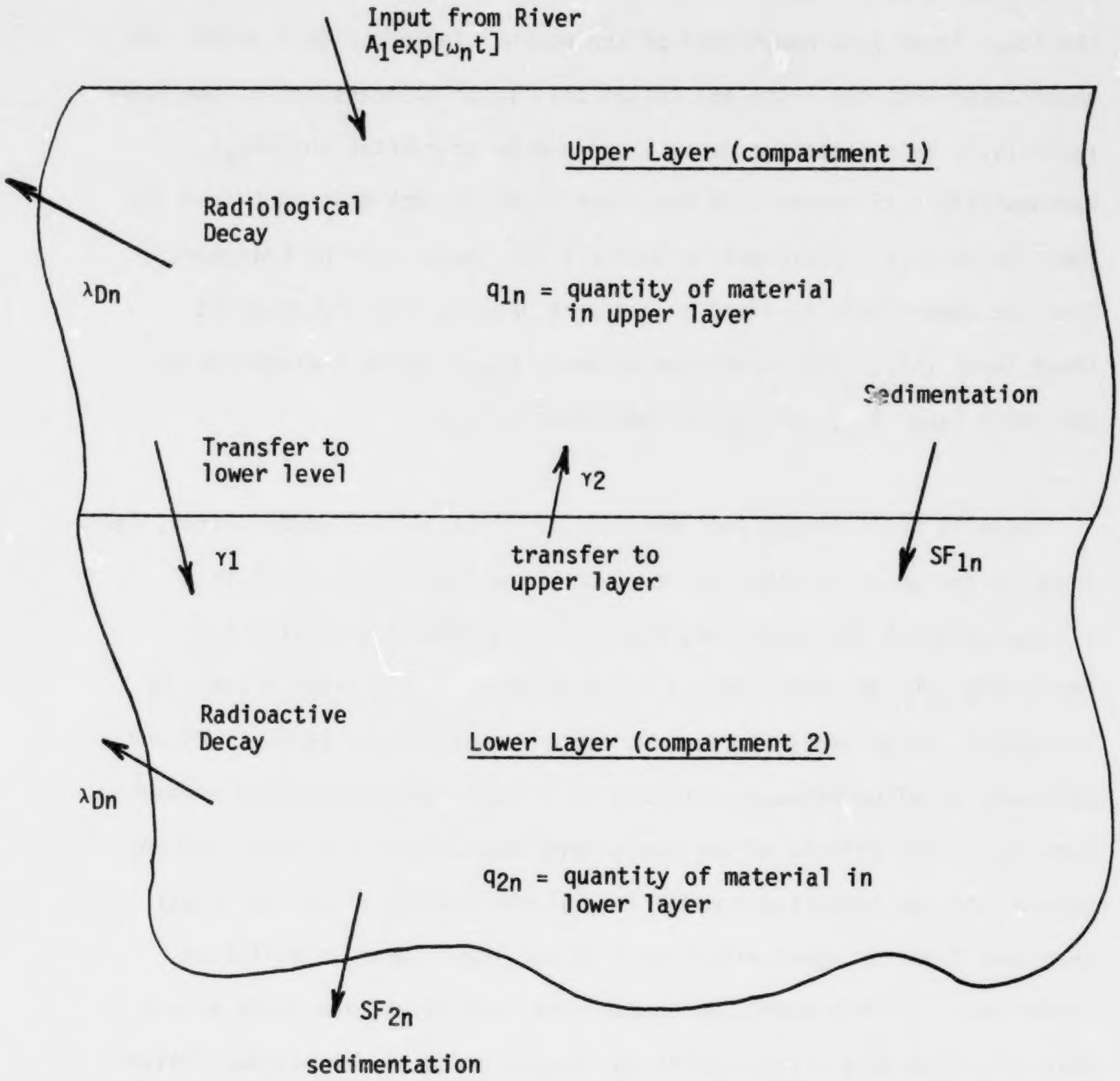
Input from River
$A_1 \exp[\omega_n t]$
Upper Layer (compartment 1)
Radiological Decay
λ_{Dn}
q_{1n} = quantity of material in upper layer
Sedimentation
Transfer to lower level
γ_2
γ_1
transfer to upper layer
SF_{1n}
Radioactive Decay
Lower Layer (compartment 2)
λ_{Dn}
q_{2n} = quantity of material in lower layer
SF_{2n}
sedimentation

Fig. 3-1. Compartment model for the ocean release mode

Compartment 1 is the upper mixed layer of the ocean and compartment 2 is the lower layer (the remainder) of the ocean. Radionuclide n enters the upper layer from the river and leaves this layer by transport to the lower layer (γ_1), by radioactive decay (λ_{Dn}) and by sedimentation (SF_{1n}). Radionuclide n is returned to the upper layer by back-transport from the lower layer (γ_2). Radionuclide n enters the lower layer by transport from the upper layer (γ_1) and by sediment passing from the upper to lower layer (SF_{1n}) and is removed by decay (λ_{Dn}), by back-transport to the upper layer (γ_2), and by sedimentation (SF_{2n}).

Several basic assumptions are made in deriving this model. First, the input to the ocean is taken to be of the form $A_1\exp(\omega_n t)$, which is assumed to equal the input into the river (equation 2.1-2) from a repository with no reductions of input by decay during river travel, by irrigation, or by sedimentation. These are conservative assumptions and were made to allow releases directly to an ocean to be evaluated without considering the effects of various removal mechanisms in a river system. Second, the two compartment model is a simplification since the actual transport from the upper mixed layer to the lower layer is diffusion controlled. In this model, we assume that both layers are fully mixed. This will lead to a discontinuity in concentration at the boundary between the two layers. Third, fish and shellfish ingestion are considered to be the only non-negligible routes of radiation uptake and exposure to humans via the ocean pathway. Fourth, all edible fish and shellfish are assumed to be taken from the upper compartment.

The steps involved in computing the environmental risk commitment from each nuclide are outlined below:

(a) Calculate the quantity of radionuclide n in the upper (mixed) layer of the ocean, q_{1n}, as a function of time, and the concentration in ocean water as a function of time.

(b) Calculate the concentration of radionuclide n in the edible fish and shellfish in the upper layer as a function of time.

(c) Apply the appropriate ingestion rates by the population for fish and shellfish to obtain the total ingestion radionuclide intake rate.

(d) Apply the appropriate ingestion fatal cancer risk factors to determine the risk commitment rate to the population from fish and shellfish consumption.

(e) Integrate the population risk commitment rate from time of arrival of radionuclides in the ocean to the desired time, t, to get the environmental risk commitment.

3.2.2 Ocean Two-Compartment Model

To calculate the concentration of radionuclide n in the upper compartment, the quantity of n in the upper compartment, q_{1n}, must be predicted as a function of time and divided by the volume, V_1, of the upper compartment. To obtain q_{1n}, we must write a system of two coupled differential equations based on the nuclide balance in each compartment as depicted in Fig. 3.2.1-1. These equations are

$$\frac{dq_{1n}}{dt} = A_1 e^{\omega_n t} + \gamma_2 q_{2n} - (\lambda_{Dn} + \gamma_1 + SF_{1n}) q_{1n} \qquad (3.2.2\text{-}1)$$

and

$$\frac{dq_{2n}}{dt} = (SF_{1n} + \gamma_1) q_{1n} - (SF_{2n} + \lambda_{Dn} + \gamma_2) q_{2n} \qquad (3.2.2\text{-}2)$$

with the associated initial conditions of

$$q_{1n} = q_{2n} = 0 \quad \text{at} \quad t = t_{Rn}.$$

After transforming variables, these coupled differential equations may be solved by methods such as successive elimination to yield q_{1n} and q_{2n}. Since q_{2n}, the radioactivity in the lower compartment, is not used in the analysis for the ocean pathway, the analytical expression is not presented. Then

$$q_{1n} = \frac{A_{2n}}{(M_{2n} - M_{1n})} \left\{ \frac{(a_{1n} - M_{2n})}{(\omega_n - M_{1n})} e^{M_{1n}(t-t_{Rn})} - \frac{(a_{1n} - M_{1n})}{(\omega_n - M_{2n})} e^{M_{2n}(t-t_{Rn})} + \frac{(M_{2n} - M_{1n})(\omega_n - b_{2n})}{(\omega_n - M_{2n})(\omega_n - M_{1n})} e^{\omega_n(t-t_{Rn})} \right\}$$

where (3.2.2-3)

$$A_{2n} = \lambda_{Ln} f_L Q_{on} e^{-\lambda_{Dn} t_{Rn}}$$

$$a_{1n} = -(\lambda_{Dn} + SF_{1n} + \gamma_1)$$

$$b_{2n} = -(\lambda_{Dn} + SF_{2n} + \gamma_2)$$

$$\omega_n = -(\lambda_{Dn} + \lambda_{Ln})$$

$$B_{3n} = -(a_{1n} + b_{2n})$$

$$C_{3n} = (a_{1n} . b_{2n}) - [\gamma_1 \gamma_2 + \gamma_2 SF_{1n}]$$

$$M_{1n} = \frac{- B_{3n} + \sqrt{B_{3n}^2 - 4C_{3n}}}{2}$$

and

$$M_{2n} = \frac{- B_{3n} - \sqrt{B_{3n}^2 - 4C_{3n}}}{2} .$$

Equation 3.2.2-3 can be used to predict the quantity of a radionuclide, n, which is uniformly mixed within the upper compartment of the ocean at any time, t, after placement of radionuclides in a waste repository. If equation 3.2.2-3 is divided by V_1, an equation to express the average concentration of nuclide n in the upper compartment of the ocean is obtained.

3.2.3 Seafood Ingestion

Ocean Fish (p=9)
Ocean Shellfish (p=10)

The equation used to calculate ERC for these pathways is

$$FHE_{np} = \frac{CF_{np} \, I_p \, P_p \, FCF_{np}}{V_1} \int_{t_{Rn}}^{t} q_{1n} \, dt'' \qquad (3.2.3\text{-}1)$$

where q_{1n} is described in equation 3.2.2-3. Equation 3.2.3-1 is integrated between the limits of t_{Rn} and t since no dose is incurred prior to time $t=t_{Rn}$. After integration and normalization by the integrated source term (equation 2.1-4), the following expression for the ERC to an exposed population due to consumption of seafood is obtained:

$$\frac{FHE_{np}}{Q_{np}} = \frac{FCF_{np}\; CF_{np}\; I_p\; P_p}{V_1} \left[\frac{\lambda_{Dn} + \lambda_{Ln}}{1 - e^{-(\lambda_{Dn} + \lambda_{Ln})(t - t_{Rn})}} \right] (COM_n) \qquad (3.2.3\text{-}2)$$

where

$$COM_n = \left(\frac{1}{M_{2n} - M_{1n}} \right) \left\{ \begin{array}{l} \dfrac{(a_{1n} - M_{2n}) \left[\exp[M_{1n}\,(t - t_{Rn})] \quad -1 \right]}{(\omega_n - M_{1n})\,(M_{1n})} \\[2ex] - \dfrac{(a_{1n} - M_{1n}) \left[\exp[M_{2n}\,(t - t_{Rn})] \quad -1 \right]}{(\omega_n - M_{2n})\,(M_{2n})} \\[2ex] + \dfrac{(M_{2n} - M_{1n})\,(\omega_n - b_{2n}) \left[\exp[\omega_n (t - t_{Rn})] \quad -1 \right]}{(\omega_n - M_{2n})\;\;(\omega_n - M_{1n})\,(\omega_n)} \end{array} \right\}. \qquad (3.2.3\text{-}3)$$

3.3 Releases to the Land Surface*

3.3.1 General Considerations

As discussed in Chapter 2, we assume that radioactive material is placed in a repository at time t=0 and that the material is brought to the surface of the earth at $t=t_L$ by some event such as exploratory drilling for resources. The release to the surface of the earth is assumed to be over a small area and over a short period of time so that the source to the land surface can be modeled as an instantaneous point source. The released material is assumed to be uniformly distributed, vertically, within the top 15 cm of soil rather than concentrated on the soil surface. The philosophy behind this assumption is based on an article by Cline (C184) where it is shown that, in the absence of tillage, phenomenon such as biological transport, contraction and expansion cycles, mass flow, and diffusion can significantly flatten expected vertical radionuclide profiles for measurements taken 25 years following soil surface contamination. The choice of a 15 cm vertical mixing depth is arbitrary and probably conservative. Cline's data indicate that enhanced mixing, due to the phenomenon listed above, might occur to soil depths of 30 to 100 cm. The 15 cm depth was chosen partly for the sake of conservatism and partly for the convenience of remaining consistent with the soil distribution assumptions chosen for the river release mode. The distribution of source material within the top 15 cm of soil is not nearly as conservative as assuming all material remains indefinitely on the earth's surface.

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*The variables used in the equations in this section are defined in the "Nomenclature" section, p. N-1 ff.

Once the initial source deposition to the soil is determined, calculations can be made to estimate the resuspension of radioactive material at this source and to predict how this material disperses in the environment. As for the river release mode, it is assumed that resuspension occurs for the radioactive material in the top 1 cm of soil. The resuspended material results in exposure to the population due to consumption of contaminated food crops, inhalation of the resuspended material, and external exposure due to ground contamination and air submersion.

3.3.2 Food Ingestion

Food crops ($p=13$)
Milk ($p=14$)
Beef ($p=15$)

For the land surface release mode, predicting uptake of radionuclides into foods and the subsequent ingestion of radioactivity by human receptors involves modelling resuspension from the ground source to air, air dispersion, and deposition to the land surface. When land surface deposition has been determined, the RI_{np} factors discussed in section 3.1.4* can be applied to quantify ingestion of radionuclides by man. The additional steps involved in determining the environmental risk commitment for food ingestion are similar to those discussed in section 3.1.4.

In calculating air concentration as a function of distance and time, we assume Nelson's (Ne 78) isotropic dispersion, which was based on annual-average atmospheric dispersion factor data for ground-level releases at 17 nuclear power reactor sites as presented in Appendix I to 10 CFR 50 (AEC73). The equation is

$$X_{in}(r,t') = \left(\frac{X}{Q'}\right)_{r_n} Q'(t') \left(\frac{r}{r_n}\right)^{-z} \qquad (3.3.2\text{-}1)$$

where

$X_{in}(r,t')$ = air concentration at point r and time t' (Ci/m^3),

$(X/Q')_{r_n}$ = atmospheric dispersion factor at the known point r_n (sec/m^3),

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*The derivation of these RI_{np} factors is discussed in more detail in Appendix C, section C.1.

$Q'(t')$ = source term from ground to air at time t' (Ci/yr)

z = "fitting" exponent to adjust shape of $X(r,t')$ curve to agree with empirical data, and

t' = time after material in repository reaches ground surface (yr).

An equation that describes $Q'(t')$ in terms of the material initially present at the ground surface is

$$Q'(t') = \lambda_R (Q_{np}/15) \exp [- (\lambda_R + \lambda_{Dn} + \lambda_{Sn}) t'] \quad . \qquad (3.3.2\text{-}2)$$

But this equation is not corrected for plume depletion during travel. A correction factor which makes a downward adjustment of the source term, $Q'(t')$, to correct for plume depletion can be expressed as

$$\frac{Q'_D(t')}{Q'(t')} = \exp \left[-\left(\frac{r}{r_d}\right)^{(2-z)} \right] \qquad (3.3.2\text{-}3)$$

where

$$r_d = \left[\frac{(2-z)\, r_n^{-z}}{2\pi v_{gn} (X/Q')_{r_n}} \right]^{\left(\frac{1}{2-z}\right)} \quad . \qquad (3.3.2\text{-}4)$$

Combining equations 3.3.2-2 and 3.3.2-3, we have

$$Q'_D(t') = \lambda_R (Q_{np}/15) \exp[-(\lambda_R + \lambda_{Dn} + \lambda_{Sn}) t'] \exp\left[-\left(\frac{r}{r_d}\right)^{(2-z)}\right] \; ; \qquad (3.3.2\text{-}5)$$

and the equation for air concentration at a point r and at time t' due to dispersion of material resuspended from the point source is (substituting $Q'_D(t')$ for $Q'(t')$ in equation 3.3.2-1)

$$X_{in}(r,t') = \left(\frac{X}{Q'}\right)_{r_n} \left(\frac{r}{r_n}\right)^{-z} \lambda_R \left(\frac{Q_{np}}{15}\right) \exp\left[-(\lambda_R+\lambda_{Dn}+\lambda_{Sn})t' - \left(\frac{r}{r_d}\right)^{(2-z)}\right]. \qquad (3.3.2\text{-}6)$$

We next estimate the deposition to the ground surface at distances away from the resuspension point by multiplying equation 3.3.2-6 by the dry deposition velocity. An equation that expresses the total deposition to the ground surface over all area and to time t' is obtained from equation 3.3.2-6 as follows:

$$DEP(\infty,t') = \int_{r=0}^{\infty} \int_{\theta=0}^{2\pi} \int_{t''=0}^{t'} X_{in}(r,t'') v_{gn} \, dt'' \, (r d\theta dr). \qquad (3.3.2\text{-}7)$$

Performing these integrations yields an expression for the total deposition to the ground surface to time t':

$$DEP(\infty,t') = DEP(t') = \frac{\lambda_R(Q_{np}/15)}{\lambda_R+\lambda_{Dn}+\lambda_{Sn}} \left[1 - \exp[-(\lambda_R+\lambda_{Dn}+\lambda_{Sn})t']\right]. \qquad (3.3.2\text{-}8)$$

To obtain the ERC for any one of the food pathways, we use a method similar to that described in section 3.1.4. The equation is

$$FHE_{np} = \left[\frac{DEP(t')}{A_r}\right] f_p \, RI_{np} \, (CP_p \, A_r) \, FCF_{np} \qquad (3.3.2\text{-}9)$$

where A_r is land area on which resuspended material is deposited, and the other terms have been previously defined or are listed in the "Nomenclature" section.

Upon cancelling the area terms, substituting equation 3.3.2-8 for DEP(t'), normalizing by the quantity of radionuclides initially released to the ground surface, Q_{np}, and redefining $t'=t-t_L$, equation 3.3.2-9 becomes

$$\frac{FHE_{np}}{Q_{np}} = \frac{f_p \, RI_{np} \, CP_p \, FCF_{np} \, \lambda_R \left[1 - e^{-(\lambda_R + \lambda_{Dn} + \lambda_{Sn})(t - t_L)}\right]}{15 \, (\lambda_R + \lambda_{Dn} + \lambda_{Sn})} . \tag{3.3.2-10}$$

We make the conservative assumption that all farm land is used to produce edible food crops; in fact, some would be used to raise non-food crops. Conversely, there will be some food crops whose roots extend below the assumed 15 cm soil root zone such that they can take up radioactivity from the soil below this depth. For these cases, our use of a λ_{Sn} based on a 15 cm soil root zone is not conservative.

3.3.3 Inhalation of Resuspended Material (p=12)

The calculation of the ERC due to inhalation of radioactive nuclides for the land surface release mode must consider the initial resuspension at the point source. In addition, deposition to the ground surface and resuspension from the ground surface away from the source must be modeled. For the reasons discussed in section 3.1.1, it is assumed that radionuclides deposited to the land surface after dispersion away from the source are uniformly mixed within the 15 cm soil root zone and that resuspension occurs for the radionuclides within the top 1 cm of soil. These assumptions maintain consistency with the methods used for treating source resuspension for the land surface release mode (see section 3.3.1) and for treating resuspension from irrigated land for the river release mode (see section 3.1.5). These assumptions are more realistic than assuming that all material remains on the ground surface indefinitely. An activity (mass) balance around a segment of ground away from the source can be depicted as shown in Fig. 3-2.

The soil surface concentration as a function of time and location is estimated so that a resuspension factor can be applied to obtain the air concentration as a function of time and location.

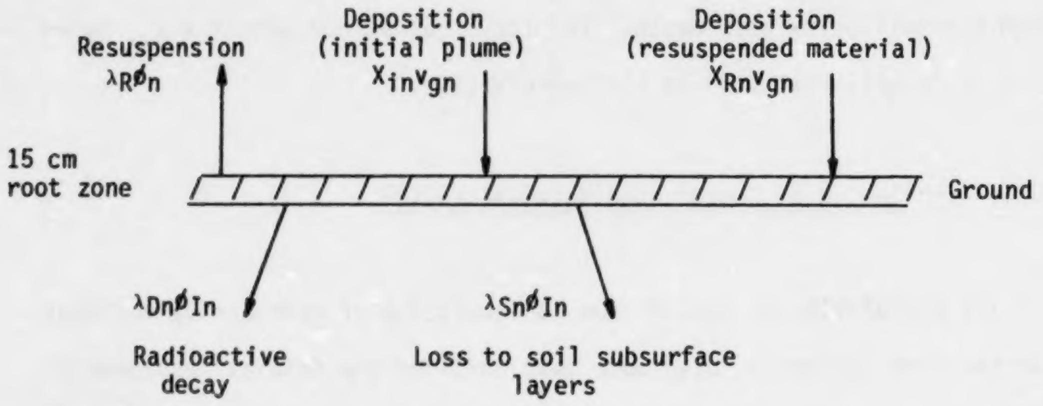
Resuspension
$\lambda_R \phi_n$
Deposition (initial plume)
$X_{in} v_{gn}$
Deposition (resuspended material)
$X_{Rn} v_{gn}$
15 cm root zone
Ground
$\lambda_{Dn} \phi_{In}$
Radioactive decay
$\lambda_{Sn} \phi_{In}$
Loss to soil subsurface layers

Fig. 3-2. Activity (mass) balance for soil element away from source

A differential equation which addresses the activity balance depicted in Fig. 3-2 is

$$\frac{d\phi_{In}}{dt'} = X_{in}v_{gn} + X_{Rn}v_{gn} - \lambda_R(\phi_{In}/15) - \lambda_{Dn}\phi_{In} - \lambda_{Sn}\phi_{In} \qquad (3.3.3\text{-}1)$$

where $\phi_n = \phi_{In}/15$, X_{in} is the air concentration of radionuclide n due to resuspension from the initial point source and subsequent dispersion and X_{Rn} is the air concentration of radionuclide n due to resuspension from the ground surface at the location of interest. The other terms have been previously defined. If the assumption is made that there is no redistribution of radioactive material due to continual deposition to and resuspension from the ground surface after equilibrium is reached, we may equate the terms $X_{Rn}v_{gn}$ and $\lambda_R\phi_{In}/15$ in equation 3.3.3-1 and write

$$\frac{d\phi_{In}}{dt'} = X_{in}\, v_{gn} - (\lambda_{Dn} + \lambda_{Sn})\, \phi_{In} . \qquad (3.3.3\text{-}2)$$

An expression for X_{in} was discussed in Section 3.3.2 (equation 3.3.2-6). If this expression for X_{in} is applied in equation 3.3.3-2 and the integration factor $\exp(\lambda_{Dn}+\lambda_{Sn})t'$ is used, the equation is solved to yield an expression for the total soil root zone inventory as a function of distance and time:

$$\phi_{In}(r,t') = v_{gn} \cdot \frac{Q_{np}}{15} \cdot \left(\frac{X}{Q'}\right)_{r_n} \cdot \left(\frac{r}{r_n}\right)^{-z} \cdot e^{-(r/r_d)^{(2-z)}} \cdot \left[e^{-(\lambda_{Dn}+\lambda_{Sn})t'} - e^{-(\lambda_R+\lambda_{Dn}+\lambda_{Sn})t'}\right].$$

(3.3.3-3)

Assuming, as discussed in Section 3.1.5, that the air concentration due to resuspension can be calculated by applying a resuspension factor to the soil surface concentration (ϕ_n, which is $\phi_{In}/15$), we have

$$X_{Rn} = RF\ \phi_{In}(r,t')/15. \qquad (3.3.3\text{-}4)$$

Then the total air concentration at a particular location and time is

$$X_n(r,t') = X_{in}(r,t') + X_{Rn}(r,t') \qquad (3.3.3\text{-}5)$$

and using equations 3.3.2-6, 3.3.3-3, and 3.3.3-4 we have

$$X_n(r,t') = \lambda_R\, Q_{np} \left(\frac{X}{Q'}\right)_{r_n} \left(\frac{r}{r_n}\right)^{-z} e^{[-(r/r_d)^{(2-z)} - \lambda_{Tn} t']} \left[0.0622 + 0.0044\, e^{\lambda_R t'}\right]$$

where $\lambda_{Tn} = \lambda_R + \lambda_{Dn} + \lambda_{Sn}$. (3.3.3-6)

Equation 3.3.3-6 yields an expression for air concentration to use in computing the ERC due to inhalation of resuspended material. The relationship to apply for calculating ERC is

$$FHE_{np} = \int_0^{t'} \int_0^{A} X_n(r,t'')\, I_B\, FCF_{np}\, PD_p\, dA_r\, dt'' \;. \qquad (3.3.3\text{-}7)$$

This equation is similar to the one derived for the inhalation pathway in Section 3.1.5. Using the expression for $X_n(r,t')$ from equation 3.3.3-6, the relationship $dA_r = r\,d\theta\,dr$, performing the area integration from $r=0$ to $r=\infty$ and $\theta=0$ to $\theta=2\pi$ (integrating over all area where people are exposed), and normalizing the result by the quantity of radionuclides released to the ground surface, Q_{np}, we have

$$\frac{FHE_{np}}{Q_{np}} = (PD_p)(RF)(I_B)(FCF_{np}) \left\{ \frac{0.0622}{\lambda_{Tn}} \left[1 - e^{-\lambda_{Tn} t'}\right] + \frac{0.0044}{\lambda_{Dn} + \lambda_{Sn}} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})t'}\right] \right\}$$

(3.3.3-8)

Redefining t' as $t-t_L$ the equation for ERC can be written

$$\frac{FHE_{np}}{Q_{np}} = (PD_p)(RF)(I_B)(FCF_{np}) \left\{ \frac{0.0622}{\lambda_{Tn}} \left[1 - e^{-\lambda_{Tn}(t-t_L)}\right] + \frac{0.0044}{\lambda_{Dn} + \lambda_{Sn}} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})(t-t_L)}\right] \right\} \tag{3.3.3-9}$$

3.3.4 External Risk Commitment-Ground Contamination (p=16)

The equation to apply in calculating ERC is

$$FHE_{np} = \int_0^{t'} \int_0^{A} \phi_{In}(r,t'')\, FCF_{np}\, SOF\, GC_{np}\, PD_p\, dA\, dt''. \tag{3.3.4-1}$$

Using the expression for $\phi_{In}(r,t')$ developed in Section 3.3.3 (equation 3.3.3-3), the relationship $dA_r = r d\theta dr$ with the integration limits $0 \leq r \leq \infty$ and $0 \leq \theta \leq 2\pi$, normalizing the result by the initial release to the ground surface, Q_{np}, and redefining t' as $t-t_L$, equation 3.3.4-1 becomes

$$\frac{FHE_{np}}{Q_{np}} = \frac{PD_p\, FCF_{np}\, SOF\, GC_{np}}{15} \left\{ \frac{1 - e^{-(\lambda_{Dn} + \lambda_{Sn})(t-t_L)}}{\lambda_{Dn} + \lambda_{Sn}} + \frac{e^{-\lambda_{Tn}(t-t_L)} - 1}{\lambda_{Tn}} \right\} \tag{3.3.4-2}$$

where λ_{Tn} was defined in Section 3.3.3.

3.3.5 External Risk Commitment-Air Submersion (p=11)

The equation for calculating ERC is

$$FHE_{np} = \int_0^{t'} \int_0^{A} X_n(r,t'')\, FCF_{np}\, SOF\, PD_p\, dA\, dt'' . \qquad (3.3.5\text{-}1)$$

Using the expression for $X_n(r,t')$ developed in Section 3.3.3 (equation 3.3.3-6), the relationship $dA = r d\theta dr$ with the integration limits $0 \leq r \leq \infty$ and $0 \leq \theta \leq 2\pi$, normalizing the result by the source term Q_{np}, and redefining t' as $t-t_L$, equation 3.3.5-1 becomes

$$\frac{FHE_{np}}{Q_{np}} = PD_p \cdot FCF_{np} \cdot SOF \cdot RF \left\{ \frac{0.0622}{\lambda_{Tn}} \left[1 - e^{-\lambda_{Tn}(t-t_L)} \right] + \frac{0.0044}{\lambda_{Dn} + \lambda_{Sn}} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})(t-t_L)} \right] \right\} \qquad (3.3.5\text{-}2)$$

3.4 Releases Due to Volcano/Meteorite Interaction*

3.4.1 General Considerations

We assume that either a volcano or a meteorite interacts with a waste repository and distributes material to the air and directly to the land surface in a small area around the volcano or meteorite. Because the interactions are violent, we assume that the material released into air would be distributed uniformly throughout the troposphere. Radionuclides released to air will affect people differently depending on whether the nuclides are in the inhalation zone over land or in the air over the ocean and on whether they deposit on land or in the ocean. We account for this by splitting the inventory released to the air into two parts: that over oceans and that over land, using the respective surface areas of the oceans and the land surface of the earth. This division of the airborne material is for calculational convenience in developing the mathematical equations for the environmental dose commitment analyses. Each curie of radionuclide which is released is divided between that quantity released directly to the land surface (f_{LL}), that released to air above land (f_{AL}), and that released to air above oceans (f_{AW}). We assume that airborne material above land remains over land and that airborne material above water remains over water.

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*The variables used in the equations in this section are defined in the nomenclature, p. N-1 ff.

3.4.2 Releases Directly to Land Surface

3.4.2.1 General Considerations

The radionuclides released directly to the land surface are conservatively assumed to be distributed in a small area around the release point. The methods used to determine vertical distribution and resuspension at the source, dispersion in the environment, and resulting ERC to the affected population are the same as those devised for the land surface pathway (Section 3.3). The equations used for releases directly to the land surface will be listed, for the sake of completeness, in the following sections.

3.4.2.2 Food Ingestion

Food Crops (p=25)
Milk (p=26)
Beef (p=27)

$$\frac{FHE_{np}}{Q_{np}} = f_p \; RI_{np} \; CP_p \; FCF_{np} \; f_{LL} \; \lambda_R \; \frac{\left[1 - e^{-\lambda_{Tn}(t-t_v)}\right]}{15\,\lambda_{Tn}} \qquad (3.4.2.2\text{-}1)$$

where f_{LL} is the fraction of the radionuclides released to the environment which go directly to land and t_v is the time after placement of radioactive material in the repository that material is released to the environment.

3.4.2.3 Inhalation of Resuspended Material (p=24)

$$\frac{FHE_{np}}{Q_{np}} = PD_p \; RF \; I_B \; FCF_{np} \; f_{LL} \left\{ \frac{0.0622}{\lambda_{Tn}} \left[1 - e^{-\lambda_{Tn}(t-t_v)}\right] + \frac{0.0044}{\lambda_{Dn} + \lambda_{Sn}} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})(t-t_v)}\right] \right\} \qquad (3.4.2.3\text{-}1)$$

3.4.2.4 External Risk Commitment-Ground Contamination (p=28)

$$\frac{FHE_{np}}{Q_{np}} = \frac{PD_p \; FCF_{np} \; SOF \; f_{LL} \; GC_{np}}{15} \left[\frac{1 - e^{-(\lambda_{Dn} + \lambda_{Sn})(t-t_v)}}{\lambda_{Dn} + \lambda_{Sn}} + \frac{e^{-\lambda_{Tn}(t-t_v)} - 1}{\lambda_{Tn}} \right]. \qquad (3.4.2.4\text{-}1)$$

3.4.2.5 External Risk Commitment-Air Submersion (p=23)

$$\frac{FHE_{np}}{Q_{np}} = SOF \quad FCF_{np} \; PD_p \; RF \quad f_{LL} \left\{ \begin{array}{l} \dfrac{0.0622}{\lambda_{Tn}} \left[1 - e^{-\lambda_{Tn}(t-t_v)}\right] \\ + \dfrac{0.0044}{\lambda_{Dn} + \lambda_{Sn}} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})(t-t_v)}\right] \end{array} \right\} \quad (3.4.2.5\text{-}1)$$

3.4.3 Releases to Air-Over-Land

3.4.3.1 General Considerations

The radionuclides released to the air over land surfaces are assumed to be distributed uniformly in a volume determined by multiplying the land surface area of the earth by the average height of the troposphere. With the material distributed in this manner a two compartment model is established, as depicted in Fig. 3-3, to predict radionuclide movement between the air and the soil for use in computing the ERC for the various pathways. The upper compartment in Fig. 3-3 is the tropospheric volume above the earth's land surface and the lower compartment is the available land layer, i.e., the layer of land containing the soil surface as an upper boundary and including the root zone or plow layer of soil. It is assumed that radionuclides enter the upper compartment at the instant a volcano or meteorite interaction releases radioactivity from the repository. No further radionuclides are introduced into the system after the initial input at t'= 0.

Radionuclides leave the upper compartment by radioactive decay (λ_{Dn}) and by transfer from air to soil ($v_{gn} . A_L / V_L$) and they reenter the

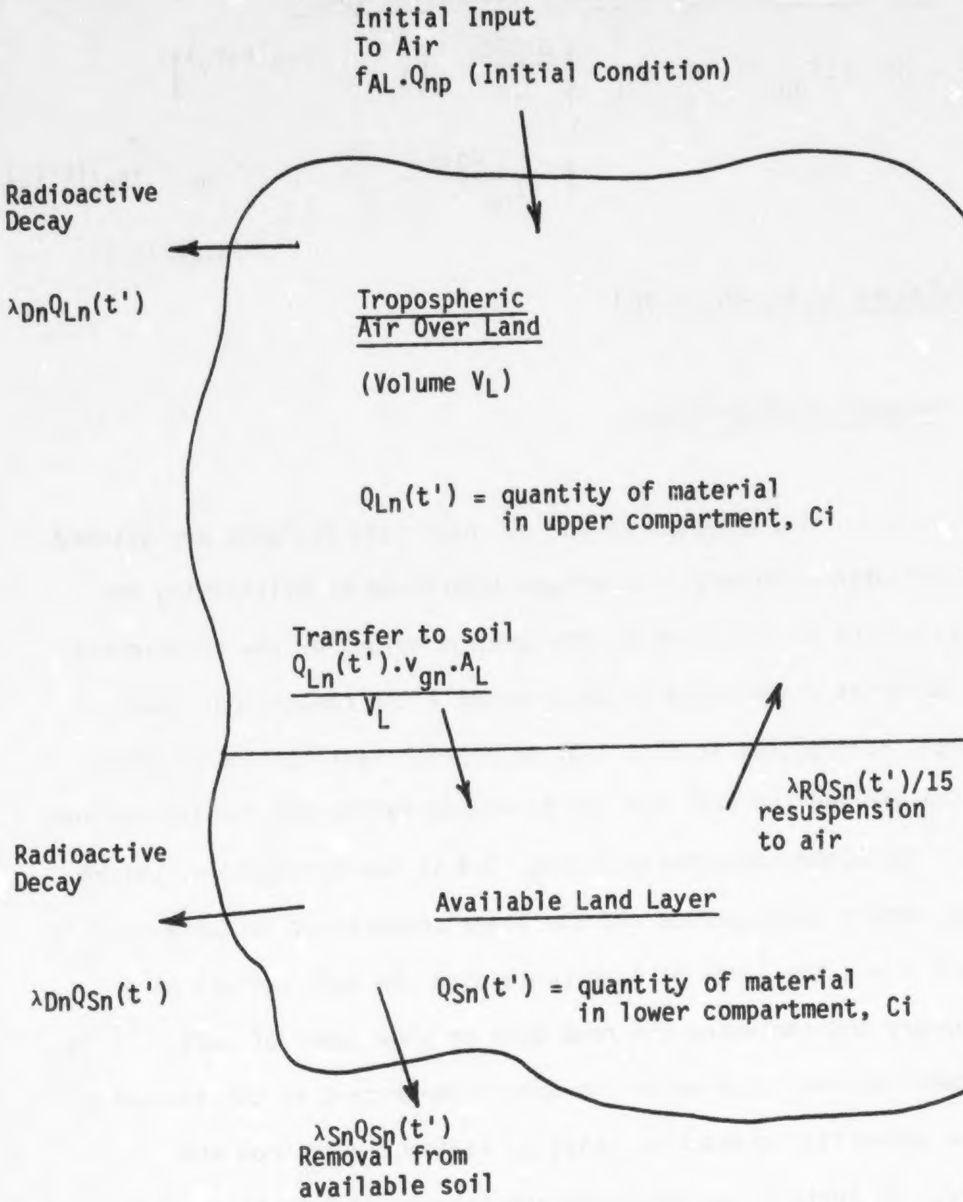
Initial Input
To Air
$f_{AL} \cdot Q_{np}$ (Initial Condition)
Radioactive Decay
$\lambda_{Dn} Q_{Ln}(t')$
Tropospheric Air Over Land
(Volume V_L)
$Q_{Ln}(t')$ = quantity of material in upper compartment, Ci
Transfer to soil
$\frac{Q_{Ln}(t') \cdot v_{gn} \cdot A_L}{V_L}$
$\lambda_R Q_{Sn}(t')/15$
resuspension to air
Radioactive Decay
Available Land Layer
$\lambda_{Dn} Q_{Sn}(t')$
$Q_{Sn}(t')$ = quantity of material in lower compartment, Ci
$\lambda_{Sn} Q_{Sn}(t')$
Removal from available soil

Fig. 3-3. Compartment model for air over land, volcano/meteorite release mode

upper compartment due to resuspension (λ_R). The assumptions controlling our resuspension calculations are the same as discussed in sections 3.1 and 3.3. When radionuclides are deposited to the ground surface, we assume that they quickly mix in the soil root zone (15 cm vertical depth) and that only the radionuclides in the top 1 cm of soil are subject to resuspension. Radionuclides enter the lower compartment by deposition from air ($v_{gn} \cdot A_L/V_L$) and are removed from the lower compartment by transfer to the unavailable soil layer (λ_{Sn}), radioactive decay (λ_{Dn}), and resuspension from the top 1 cm of soil layer to air (λ_R). The radionuclide balance equations which can be written for this two compartment system are discussed in the next section.

3.4.3.2 Air-Above-Land: Two Compartment Model

To obtain the concentration of radionuclide n in the upper (air) compartment, the quantity of n in the upper compartment, $Q_{Ln}(t')$, must be predicted as a function of time and divided by the volume, V_L, of the upper air compartment. Similarly, to obtain the soil root zone inventory of radionuclide n for the lower compartment, $\phi_{In}(t')$, the quantity of n in the lower compartment, $Q_{Sn}(t')$, must be predicted as a function of time and divided by the surface area, A_L, for the lower compartment. To obtain $Q_{Ln}(t')$ and $Q_{Sn}(t')$, a system of two coupled differential equations, based on the nuclide balance shown in Fig. 3-3, is written and solved. These equations are:

$$\frac{dQ_{Ln}}{dt} = - \lambda_{Dn} Q_{Ln} - \frac{v_{gn} A_L}{V_L} Q_{Ln} + \lambda_R Q_{Sn}/15 \qquad (3.4.3.2\text{-}1)$$

and

$$\frac{dQ_{Sn}}{dt} = \frac{v_{gn}\ A_L}{V_L}\ Q_{Ln} - \lambda_{Dn}\ Q_{Sn} - \lambda_{Sn}\ Q_{Sn} - \lambda_R\ Q_{Sn}/15, \qquad (3.4.3.2\text{-}2)$$

and the initial conditions for our model are

$$Q_{Ln}(0) = f_{AL}\ Q_{np}$$

and

$$Q_{Sn}(0) = 0 \qquad \text{at } t' = 0\ .$$

After a transformation of variables, these coupled differential equations may be solved to yield Q_{Ln} and Q_{Sn} as follows:

$$Q_{Ln}(t') = \frac{f_{AL} \cdot Q_{np}}{M_{6n} - M_{5n}} \left[(M_{6n} - a_{5n})\ e^{M_{5n}t'} + (a_{5n} - M_{5n})\ e^{M_{6n}t'} \right] \qquad (3.4.3.2\text{-}3)$$

$$Q_{Sn}(t') = \frac{15\ f_{AL}\ Q_{np}\ (M_{5n} - a_{5n})(M_{6n} - a_{5n})}{\lambda_R\ (M_{6n} - M_{5n})} \left[e^{M_{5n}t'} - e^{M_{6n}t'} \right] \qquad (3.4.3.2\text{-}4)$$

where

$$a_{5n} = -\left(\lambda_{Dn} + \frac{v_{gn}}{h_a}\right)$$

$$b_{6n} = -\left(\lambda_{Dn} + \lambda_{Sn} + \lambda_R/15\right)$$

$$B_{4n} = -\left(a_{5n} + b_{6n}\right)$$

$$C_{4n} = a_{5n}\ b_{6n} - \frac{v_{gn}\ \lambda_R}{15\ h_a}$$

$$M_{5n} = -\frac{B_{4n} + \sqrt{B_{4n}^2 - 4C_{4n}}}{2}$$

$$M_{6n} = -\frac{B_{4n} - \sqrt{B_{4n}^2 - 4C_{4n}}}{2}\ .$$

Now, using equations 3.4.3.2-3 and 3.4.3.2-4, the air and ground concentrations of radionuclides as a function of time can be calculated.

To compute the air concentration in the upper compartment as a function of time, equation 3.4.3.2-3 is divided by the volume of the upper compartment, which is $V_L = A_L . h_A$, to yield

$$X_n(t') = \frac{f_{AL} . Q_{np}}{A_L . h_A (M_{6n} - M_{5n})} \left[(M_{6n} - a_{5n}) e^{M_{5n} t'} + (a_{5n} - M_{5n}) e^{M_{6n} t'} \right] \quad (3.4.3.2\text{-}5)$$

The soil root zone inventory for the lower compartment as a function of time is obtained by dividing equation 3.4.3.2-4 by the surface area, A_L, of the lower compartment to yield

$$\phi_{In}(t') = \frac{15 f_{AL} . Q_{np} (M_{5n} - a_{5n})(M_{6n} - a_{5n})}{\lambda_R (M_{6n} - M_{5n}) A_L} \left[e^{M_{5n} t'} - e^{M_{6n} t'} \right] \quad (3.4.3.2\text{-}6)$$

The equations generated above are used for the ERC pathway models discussed below.

3.4.3.3. Food Ingestion

Food crops (p=19)
Milk (p=20)
Beef (p=21)

Methods similar to those discussed in sections 3.1 and 3.3 are employed in calculating the ERC due to food ingestion. The derivation of the values for RI_{np} for food crops is discussed in detail in Appendix C. The deposition to the ground surface due to the radioactive material originally distributed in the air above the land surface (neglecting resuspension) is calculated and applied in conjunction with the values of RI_{np}. Since the RI_{np} values were determined without considering resuspension, we must compute the radionuclide flux to ground as a function of time for the radionuclides originally dispersed into the air at t'=0. Referring to Fig. 3-3, the flux of nuclide n to ground as a function of time, $F'_n(t')$, can be calculated as

$$F'_n(t') = \frac{Q_{Ln}(t') \cdot v_{gn}}{V_L} \qquad (3.4.3.3\text{-}1)$$

where, for these three pathways, $Q_{Ln}(t')$ is determined using equation 3.4.3.2-3 with λ_R set equal to 0. Setting λ_R=0 will yield $Q_{Ln}(t')$ based only on material originally dispersed in the air and neglects resuspension altogether. To obtain $F_n(t')$, the quantity of radionuclide n deposited per unit area from the time material was originally dispersed into the air (t'=0) to time t', we integrate as follows:

$$F_n(t') = \int_0^{t'} F'_n(t'')dt'' = \int_0^{t'} \frac{f_{AL} \cdot Q_{np}}{(M_{6n} - M_{5n})} \left[(M_{6n} - a_{5n})\, e^{M_{5n}t''} + (a_{5n} - M_{5n})\, e^{M_{6n}t''} \right] \frac{v_{gn}}{V_L}\, dt'' .$$

(3.4.3.3-2)

After integrating, the result is

$$F_n(t') = \frac{f_{AL} \cdot Q_{np} \cdot v_{gn}}{(M_{6n} - M_{5n}) \cdot V_L} \left[\frac{(M_{6n} - a_{5n})}{M_{5n}} \left(e^{M_{5n}t'} - 1\right) + \frac{(a_{5n} - M_{5n})}{M_{6n}} \left(e^{M_{6n}\,t'} - 1\right) \right]$$

(3.4.3.3-3)

which has units of Ci deposited per m^2. The ERC for these pathways is derived in a manner similar to the methods applied in Section 3.1.4 and may be computed using the following equation:

$$FHE_{np} = F_n(t') \quad f_p \quad RI_{np} \quad FCF_{np} \; CP_p \quad A_L \quad . \qquad (3.4.3.3\text{-}4)$$

After substituting equation 3.4.3.3-3 for $F_n(t')$, normalizing by the original total release, Q_{np}, substituting $V_L = A_L . h_A$, and making the substitution $t' = t - t_v$, we have

$$\frac{FHE_{np}}{Q_{np}} = \frac{f_{AL} \; v_{gn} \; FCF_{np} \; RI_{np} \; f_p \; CP_p}{h_A} \left\{ \begin{array}{l} \frac{(M_{6n} - a_{5n})}{M_{5n}(M_{6n} - M_{5n})} \left[e^{M_{5n}(t-t_v)} - 1 \right] \\ + \frac{(a_{5n} - M_{5n})}{M_{6n}(M_{6n} - M_{5n})} \left[e^{M_{6n}(t-t_v)} - 1 \right] \end{array} \right\}$$

(3.4.3.3-5)

or, defining $COML_n$ as:

$$COML_n = \frac{(M_{6n} - a_{5n})}{M_{5n}(M_{6n} - M_{5n})} \left[e^{M_{5n}(t-t_v)} - 1 \right] + \frac{(a_{5n} - M_{5n})}{M_{6n}(M_{6n} - M_{5n})} \left[e^{M_{6n}(t-t_v)} - 1 \right]$$

(3.4.3.3-6)

$$\frac{FHE_{np}}{Q_{np}} = \frac{f_{AL} \; v_{gn} \; FCF_{np} \; RI_{np} \; f_p \; CP_p \; COML_n}{h_A} \quad . \qquad (3.4.3.3\text{-}7)$$

We make the conservative assumption that all farm land is used to produce edible food crops; in fact, some would be used to raise non-food crops. Conversely, there will be some food crops whose roots extend below the assumed 15 cm root zone such that they can take up radioactivity from the soil below this depth. For these cases, our use of a λ_{Sn} based on a 15 cm soil root zone is not conservative.

3.4.3.4 Inhalation of Dispersed and Resuspended Material (p=17)

In considering the ERC due to inhalation of dispersed and resuspended material an equation similar to the one derived in Section 3.1.5 was used, which is

$$FHE_{np} = \left[\int_0^{t'} X_n(t'')\, dt'' \right] I_B\, FCF_{np}\, PD_p\, A_L \quad . \qquad (3.4.3.4\text{-}1)$$

Since the equation for $X_n(t')$ has already been described in equation 3.4.3.2-5, it can be substituted into equation 3.4.3.4-1. After performing the indicated integration, normalizing by the release of radionuclide n at t'=0 and replacing t' by $t-t_v$, we have

$$\frac{FHE_{np}}{Q_{np}} = \frac{f_{AL}\; I_B\; FCF_{np}\; PD_p}{h_A}\; COML_n \quad . \qquad (3.4.3.4\text{-}2)$$

3.4.3.5 External Risk Commitment-Ground Contamination (p=22)

The equation to use in deriving an expression for the ERC for contaminated ground is

$$FHE_{np} = \left[\int_0^{t'} \phi_{In}(t'')\, dt'' \right] FCF_{np}\; PD_p\; A_L\; SOF\;\; GC_{np} \quad . \qquad (3.4.3.5\text{-}1)$$

Equation 3.4.3.2-6 is used to express $\phi_{In}(t')$. After the equation for FHE_{np} is integrated and normalized by the initial release from the repository, Q_{np}, and t' is redefined as $t-t_v$, the ERC equation becomes

$$\frac{FHE_{np}}{Q_{np}} = 15f_{AL}FCF_{np}SOF\ GC_{np}PD_{p}\ \frac{(M_{5n}-a_{5n})(M_{6n}-a_{5n})}{\lambda_R(M_{6n}-M_{5n})}\left[\frac{[e^{M_{5n}(t-t_v)}-1]}{M_{5n}}-\frac{[e^{M_{6n}(t-t_v)}-1]}{M_{6n}}\right] \tag{3.4.3.5-2}$$

3.4.3.6 External Risk Commitment-Air Submersion (p=18)

For air submersion external risk commitments, the ERC equation is written as

$$FHE_{np} = \left[\int_0^{t'} X_n(t'')\ dt''\right] FCF_{np}\ PD_p\ A_L\ SOF\ . \tag{3.4.3.6-1}$$

Equation 3.4.3.2-5 can be substituted for $X_n(t')$. After the equation for FHE_{np} is integrated and normalized by the initial release from the repository, Q_{np}, and t' is redefined as $t-t_v$, the ERC equation becomes

$$\frac{FHE_{np}}{Q_{np}} = \frac{f_{AL}\ FCF_{np}\ PD_p\ SOF\ \ COML_n}{h_A}\ . \tag{3.4.3.6-2}$$

3.4.4 Releases to Air-Over-Oceans

3.4.4.1 General Considerations

The radionuclides released to the air over the oceans are assumed to be distributed uniformly in a volume determined by multiplying the earth's ocean area by the average height of the troposphere. With the material distributed in this manner a three compartment model is established, as shown in Fig. 3-4, to describe radionuclide movement between the air and the two ocean compartments. Using this model, concentrations of radionuclides can be determined as a function of time in the air and in each of the two ocean compartments. These concentrations can be used in estimating the ERC for the pathways considered in this section.

The upper compartment (Compartment 1) in Fig. 3-4 is the tropospheric volume above the earth's oceans. The middle compartment (Compartment 2) is the top compartment of the ocean and the lower compartment (Compartment 3) is the bottom compartment of the ocean.

We assume that radionuclides enter the air (Compartment 1) at the instant a volcano or meteorite interaction releases radioactivity from the repository and that no additional radioactivity is injected into the system after that. Radionuclides leave the air compartment by radioactive decay (λ_{Dn}) and by deposition into the ocean ($v_{gn}A_W/V_{AW}$). Radionuclides enter Compartment 2 by deposition from the air ($v_{gn}A_W/V_{AW}$) and by transfer from Compartment 3 (γ_2). Radionuclides leave Compartment 2 by radioactive decay (λ_{Dn}) and by diffusion transfer (γ_1) and sedimentation transfer (SF_{1n}) to Compartment 3. Radionuclides enter Compartment 3 by diffusion transfer (γ_1) and sedimentation transfer (SF_{1n}) from the upper ocean compartment and leave this compartment by radioactive decay (λ_{Dn}), sedimentation to the ocean floor (SF_{2n}), and transfer to the upper ocean compartment (γ_2). The differential equations which can be solved for radionuclide inventory in this three compartment system are discussed in the next section.

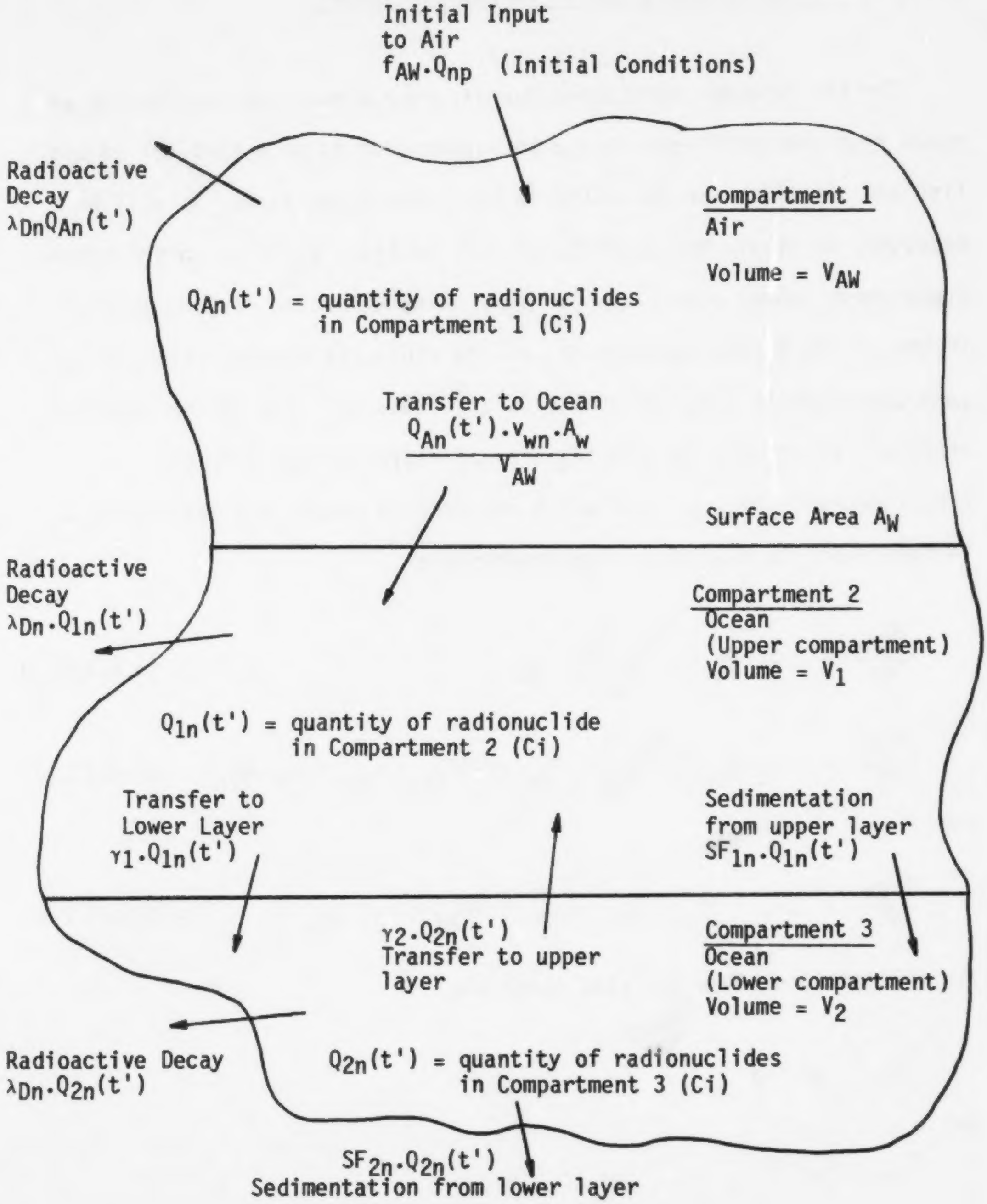
Initial Input
to Air
$f_{AW} \cdot Q_{np}$ (Initial Conditions)
Radioactive
Decay
$\lambda_{Dn} Q_{An}(t')$
Compartment 1
Air
Volume = V_{AW}
$Q_{An}(t')$ = quantity of radionuclides in Compartment 1 (Ci)
Transfer to Ocean
$\frac{Q_{An}(t') \cdot v_{wn} \cdot A_w}{V_{AW}}$
Surface Area A_W
Radioactive
Decay
$\lambda_{Dn} \cdot Q_{1n}(t')$
Compartment 2
Ocean
(Upper compartment)
Volume = V_1
$Q_{1n}(t')$ = quantity of radionuclide in Compartment 2 (Ci)
Transfer to
Lower Layer
$\gamma_1 \cdot Q_{1n}(t')$
Sedimentation
from upper layer
$SF_{1n} \cdot Q_{1n}(t')$
$\gamma_2 \cdot Q_{2n}(t')$
Transfer to upper
layer
Compartment 3
Ocean
(Lower compartment)
Volume = V_2
Radioactive Decay
$\lambda_{Dn} \cdot Q_{2n}(t')$
$Q_{2n}(t')$ = quantity of radionuclides in Compartment 3 (Ci)
$SF_{2n} \cdot Q_{2n}(t')$
Sedimentation from lower layer

Fig. 3-4. Compartment model for air-over-ocean, volcano/meteorite release mode

3.4.4.2 Air-Above-Water: Three Compartment Model

The ERC pathways considered for air-over-oceans are consumption of ocean fish and shellfish. Since the assumption is made that all edible fish and shellfish are harvested in the upper ocean layer, it will be necessary to obtain the quantity of each nuclide, $Q_{1n}(t')$, in the middle compartment (upper ocean layer). This quantity can be divided by the volume of the middle compartment, V_1, to yield the concentration in the zone where edible fish and shellfish are produced. The differential equations which describe the transfer of radionuclides between compartments in Fig. 3-4 and which are used to obtain the quantities of radionuclides in the three compartments are

$$\frac{dQ_{An}}{dt'} = -(\lambda_{Dn} + \frac{v_{wn} A_w}{V_{AW}}) Q_{An} \qquad (3.4.4.2\text{-}1)$$

$$\frac{dQ_{1n}}{dt'} = \gamma_2 Q_{2n} + \frac{v_{wn} A_w}{V_{AW}} Q_{An} - (SF_{1n} + \lambda_{Dn} + \gamma_1) Q_{1n} \qquad (3.4.4.2\text{-}2)$$

and

$$\frac{dQ_{2n}}{dt'} = (\gamma_1 + SF_{1n}) Q_{1n} - (\lambda_{Dn} + SF_{2n} + \gamma_2) Q_{2n} \quad . \qquad (3.4.4.2\text{-}3)$$

The initial conditions for this model are

$$Q_{An} = f_{AW} Q_{np}$$

and

$$Q_{1n} = Q_{2n} = 0 \qquad \text{at } t' = 0 \; .$$

The procedure used to solve these differential equations is to integrate equation 3.4.4.2-1 directly and then use the resulting equation for Q_{An} in the solution of equations 3.4.4.2-2 and 3.4.4.2-3, which are coupled differential equations. The resulting equations for Q_{An} and Q_{1n} are

$$Q_{An}(t') = f_{AW}\, Q_{np}\, e^{-(\lambda_{Dn} + \lambda_{wn})\, t'} \qquad (3.4.4.2\text{-}4)$$

$$Q_{1n}(t') = \frac{A_{2n}}{(M_{2n}-M_{1n})}\left[\frac{(a_{1n}-M_{2n})}{(\omega_n-M_{1n})} e^{M_{1n}t'} - \frac{(a_{1n}-M_{1n})}{(\omega_n-M_{2n})} e^{M_{2n}t'} + \frac{(M_{2n}-M_{1n})(\omega_n-b_{2n})}{(\omega_n-M_{2n})(\omega_n-M_{1n})} e^{\omega_n t'}\right]$$

$$(3.4.4.2\text{-}5)$$

where

$$A_{2n} = \frac{v_{wn}\, f_{AW}\, Q_{np}}{h_A}$$

$$\omega_n = -(\lambda_{Dn} + \lambda_{wn})$$

and a_{1n}, b_{2n}, B_{3n}, C_{3n}, M_{1n} and M_{2n} are as defined in Section 3.2.2. $Q_{2n}(t')$ is not needed in this analysis, so the equation for $Q_{2n}(t')$ is not presented.

The concentration of radionuclides in the upper compartment of the ocean as a function of time can be computed by dividing equation 3.4.4.2-5 by V_1, the volume of the upper ocean compartment, which yields

$$X_{1n}(t') = \frac{A_{2n}}{(M_{2n}-M_{1n})V_1}\left[\frac{(a_{1n}-M_{2n})}{(\omega_n-M_{1n})} e^{M_{1n}t'} - \frac{(a_{1n}-M_{1n})}{(\omega_n-M_{2n})} e^{M_{2n}t'} + \frac{(M_{2n}-M_{1n})(\omega_n-b_{2n})}{(\omega_n-M_{2n})(\omega_n-M_{1n})} e^{\omega_n t'}\right]$$

$$(3.4.4.2\text{-}6)$$

3.4.4.3 Seafood Ingestion

Ocean Fish (p=29)
Ocean Shellfish (p=30)

The equation used to calculate ERC for these pathways is:

$$FHE_{np} = \frac{CF_{np}\ I_p\ P_p\ FCF_{np}}{V_1} \int_0^{t'} Q_{1n}\ (t'')dt'' \qquad (3.4.4.3\text{-}1)$$

where Q_{1n} is described by equation 3.4.4.2-5. After performing the integration, normalizing by the total release, Q_{np}, and making the substitution $t'=t-t_v$, we have

$$\frac{FHE_{np}}{Q_{np}} = \frac{CF_{np}\ I_p\ P_p\ FCF_{np}\ v_{wn}\ f_{AW}}{h_A\ V_1}\ COMO_n \qquad (3.4.4.3\text{-}2)$$

where

$$COMO_n = \frac{1}{M_{2n}-M_{1n}} \left\{ \begin{array}{l} \dfrac{(a_{1n}-M_{2n})[\ e^{M_{1n}(t-t_v)} -1]}{(\omega_{sn}-M_{1n})M_{1n}} \\ -\ \dfrac{(a_{1n}-M_{1n})[\ e^{M_{2n}(t-t_v)} -1]}{(\omega_{sn}-M_{2n})M_{2n}} \\ +\ \dfrac{(M_{2n}-M_{1n})(\omega_{sn}-b_{2n})[\ e^{\omega_{sn}(t-t_v)} -1]}{(\omega_{sn}-M_{2n})(\omega_{sn}-M_{1n})\omega_{sn}} \end{array} \right\}. \qquad (3.4.4.3\text{-}3)$$

3.5 Special Calculations for C-14 Environmental Risk Commitment

The pathway models described in Sections 3.1 through 3.4 are used for all pathway ERC calculations for all nuclides except carbon-14. Unlike the other radionuclides considered in these analyses, stable carbon constitutes a significant fraction of the elemental composition of the human body and man's diet. Transport processes through the different environmental pathways and within plants, animals, and man, that apply to trace quantities of radionuclides do not necessarily apply to radionuclides, such as C-14, where the corresponding stable elements are present in such quantities that saturation effects are significant (Mo79). Atmospheric releases of C-14 as carbon dioxide can be evaluated using a diffusion-type model of the carbon cycle developed by Killough (Ki77). It seems clear that this model is the correct calculational procedure to use for releases for the volcano/meteorite release mode where it is assumed that high-temperatures would cause carbon releases to be oxidized to carbon dioxide. To our knowledge, models are not available to explicitly treat the ERC calculations for C-14 released to water, land surfaces, or air in a chemical form other than carbon dioxide. We performed some preliminary calculations using various plausible models and assumptions. Our literature review indicated that the chemical form of C-14 released in the water and land surface release modes was not well known. Also, the rate of oxidation to carbon dioxide of other chemical forms of C-14 over the extensive integration period is not known for these release modes. Considering all these uncertainties, we concluded that the most prudent course was to use the Killough carbon dioxide model for all four release modes, realizing that this probably leads to conservative estimates of the ERC for the water and land release modes.

The environmental risk commitment for C-14 is obtained by calculating the total body environmental dose commitment (EDC) and multiplying by a fatal cancer risk conversion factor. Values of the total body environmental dose commitment per curie of C-14 released to the atmosphere have been calculated by Fowler (Fo79) using the Killough model (Ki77). It is estimated that the ingestion pathway contributes 99 percent of the carbon-14 environmental dose commitment (Fo76); however, we assume that the ingestion pathways contribute 100 percent for purposes of computational convenience. For estimating the environmental dose commitment, a cubic spline was fit to Fowler's curve of worldwide EDC to the total body per curie release versus time after release. This cubic spline procedure yields the following equations for worldwide total body EDC vs time after C-14 placement in a repository:

for $10 \leq t-t_v < 100$ yr:

$$DTB_{14} = \exp \left[\begin{array}{l} 1.920 + 0.651[\ln(t-t_v) - 2.303] \\ -0.04485[\ln(t-t_v) - 2.303]^2 + 0.01109[\ln(t-t_v) - 2.303]^3 \end{array} \right] \qquad (3.5\text{-}5)$$

for $100 \leq t-t_v < 1{,}000$ yr:

$$DTB_{14} = \exp \left[\begin{array}{l} 3.316 + 0.6208[\ln(t-t_v) - 4.605] \\ +0.03172[\ln(t-t_v) - 4.605]^2 - 0.01056[\ln(t-t_v) - 4.605]^3 \end{array} \right] \qquad (3.5\text{-}6)$$

for $1{,}000 \leq t-t_v < 7{,}000$ yr:

$$DTB_{14} = \exp \left[\begin{array}{l} 4.785 + 0.5988[\ln(t-t_v) - 6.908] \\ -0.04126[\ln(t-t_v) - 6.908]^2 + 0.004198[\ln(t-t_v) - 6.908]^3 \end{array} \right] \qquad (3.5\text{-}7)$$

for $7{,}000 \leq t-t_v < 10{,}000$ yr:

$$DTB_{14} = \exp \left[\begin{array}{l} 5.825 + 0.4859[\ln(t-t_v) - 8.8537] \\ -0.01675[\ln(t-t_v) - 8.8537]^2 - 0.1544[\ln(t-t_v) - 8.8537]^3 \end{array} \right] \qquad (3.5\text{-}8)$$

for $10{,}000 \le t-t_v < 40{,}000$ yr:

$$DTB_{14} = \exp\left[\begin{array}{l} 5.989 + 0.4153[\ln(t-t_v) - 9.2103] \\ -0.1817[\ln(t-t_v) - 9.2103]^2 + 0.02385[\ln(t-t_v) - 9.2103]^3 \end{array}\right] \quad (3.5\text{-}9)$$

for $40{,}000 \le t-t_v < 100{,}000$ yr:

$$DTB_{14} = \exp\left[\begin{array}{l} 6.279 + 0.04890[\ln(t-t_v) - 10.597] \\ -0.08246[\ln(t-t_v) - 10.597]^2 + 0.04059[\ln(t-t_v) - 10.597]^3 \end{array}\right] \quad (3.5\text{-}10)$$

for $t-t_v \ge 100{,}000$ yr:

$$DTB_{14} = 537.0 \quad (3.5\text{-}11)$$

The environmental risk commitment is obtained by multiplying the total body environmental dose commitment, as obtained from equations 3.5-5 through 3.5-11, by the fatal cancer risk factor of 1.46E-4 fatal cancers per total body man-rem* as given by Fowler (Fo79). The equation is

$$\frac{FHE_{np}}{Q_{np}} = CARCAN \cdot DTB_{14} \quad (3.5\text{-}12)$$

This equation expresses the total environmental risk commitment for all pathways within a release mode and is not applied separately for each pathway. Equation 3.5-12 can be multiplied by f_{LL}, f_{AL}, and f_{AW} to estimate the C-14 ERC for the volcano/meteorite release mode for releases directly to land, releases to air over land, and releases to air over water.

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*This C-14 fatal cancer risk factor is less than would be used for most other radionuclides because a significant percentage of the total body dose from C-14 is to adipose tissue and is not effective in producing cancer (Fo79).

The C-14 dosimetry and risk information upon which our analysis is based (Fo79), estimates C-14 fatal cancer risks using total body environmental dose-equivalent commitment and a fatal cancer risk per unit total body dose-equivalent conversion factor. If we were to revise Fowler's analysis, we would use our latest RADRISK data (not available when Fowler performed his analysis) and compute an effective environmental dose-equivalent commitment and apply a fatal cancer risk per unit effective dose-equivalent conversion factor. We estimate that if our newer data had been applied, the fatal cancer risk per curie of C-14 released to the accessible environment would have increased by a factor of approximately 1.5. However, due to the rounding of the calculated radionuclide release limits in preparing Table 1 of 40 CFR 191 (EPA85c), the Table 1, C-14 release limit would not change.

Chapter 4: METHODS FOR DERIVATION OF FATAL CANCER RISK CONVERSION FACTORS*

Fatal cancer risk conversion factors are applied in each of the pathway equations discussed in Chapter 3. In some cases, risk conversion factors for daughter products are added to those for the parent** as an approximate method to account for ingrowth of daughter products during environmental transport. The methods used to obtain the risk conversion factors and to account for daughter ingrowth during environmental transport are discussed in this section.

4.1 Fatal Cancers Risk Conversion Factors

For each of the 30 pathways, either internal doses occur due to inhalation or ingestion of radionuclides or external doses are delivered due to ground contamination or air submersion and fatal cancer risk conversion factors are needed to use with the equations developed in Chapter 3. The computer code used by EPA to calculate dose and risk conversion factors is RADRISK (Du84, Su81, Du80). RADRISK calculates the radiation dose and risk resulting from an annual unit intake of a given radionuclide or the risk resulting from external exposure to a unit concentration of radionuclide in air or on the ground surface. Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the exposure associated with a specific source.

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*The variables used in this chapter are defined in the "Nomenclature" section, p. N-1 ff.

**In these instances, the sum of the risk factors is reported for the parent.

Internal exposures occur when radioactive material is inhaled or ingested. The RADRISK code implements contemporary dosimetric models to estimate the dose rates at various times to specified reference organs in the body from inhaled or ingested radionuclides. The dosimetric methods in RADRISK are adapted from those of the INREM II code (Ki78b), based primarily on models recommended by the International Commission on Radiological Protection (ICRP79) and the National Radiation Protection Board (Ad78). In some instances, input parameter values have been adjusted to be more representative of the U.S. population as a whole rather than an occupationally exposed group (Su81). The principal qualitative difference is that RADRISK computes dose rates to specified organs separately for high and low linear energy transfer (LET) radiations, whereas INREM II calculated the committed dose equivalent to specified organs.

Dose rates to organs of an individual immersed in contaminated air or standing on a contaminated ground surface are computed by the DOSFACTER computer code of Kocher (Ko81c). These calculations assume that the radionuclide concentration is uniform throughout an infinite volume of air or area of ground surface, and that the exposed individual is standing on the ground surface. Since only photons penetrate the body sufficiently to deliver a significant dose to internal organs, only doses from photon radiation are considered for the external exposure pathways.

Our fatal cancer risk conversion factors (derived using the RADRISK computer code) are generally based on fatal cancer risk data given in BEIR

3 (NAS80). The dose response functions applied in this report are linear for both low and high-LET risk estimates*. To project the number of fatalities due to leukemia and bone cancer the Agency uses an absolute risk model, a minimum induction period of two years, and a 25 year expression period. To estimate the number of fatalities due to other cancers, the Agency uses the arithmetic average of absolute and relative risk projection models. For these cancers, we assume a 10-year minimum induction period and lifetime expression of radiation induced cancer. For high-LET risk estimates, we consider the risk from high-LET (alpha particle) radiation to be eight times that for low-LET radiation to the same tissue except for bone cancer, where the high-LET risk coefficient is twenty times the low-LET value. A fuller discussion is included in Appendix G, Section G.3.

An important feature of our methodology is the use of actuarial life tables to account for the time dependence of the radiation insult and to allow for competing risks of death in the estimation of risk due to radiation exposure. A life table consists of data describing age-specific mortality rates from all causes of death for a given population. This information is derived from data obtained on actual mortality rates in a real population; mortality data for the U.S. population during the years 1969-1971 are used throughout this study.

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*We have applied the BEIR-III (NAS80) linear dose response function in computing the risk factors listed in Table 4-2 and in computing the fatal cancers per curie release to the accessible environment listed in Chapter 6. However, in Appendix G, we have included discussions of both the BEIR-III linear and the linear quadratic dose response functions for cancer induction from low-LET radiation. This has been done to show a comparison between the results obtained using the two models.

The use of life tables in studies of risk due to low-level radiation exposure is important because of the time delay inherent in radiation risk. After a radiation dose is received, there is a minimum induction period (latency period) of several years before a cancer is clinically observed. Following the latency period, the probability of occurrence of a cancer during a given year is assumed to be constant for a specified period, called a plateau period. The length of both the latency and plateau periods depends upon the type of cancer.

During or after radiation exposure, a potential cancer victim may experience years of life in which he or she is continually exposed to risk of death from causes other than incremental radiation exposure. Hence, some individuals will be lost from the population due to competing causes of death, and are not victims of incremental radiation-induced cancer.

We assume that each member of the hypothetical cohort is exposed to a specified activity of a given radionuclide. In the analysis, each member of the cohort annually inhales or ingests 1 pCi of the nuclide, or is exposed to a constant external concentration of 1 pCi/cc in air or 1 pCi/cm^2 on ground surfaces. Since the models used in RADRISK are linear, these results may be scaled to evaluate other exposure conditions. The cohort consists of an initial population of 100,000 persons, all of whom are simultaneously liveborn. In the scenario employed, the radiation exposure is assumed to begin at birth and continue throughout the entire lifetime of each individual.

No member of the cohort lives more than 110 years. The span from 0 to 110 years is divided into nine age intervals, and dose rates to specified organs at the midpoints of the age intervals are used as estimates of the annual dose during the age interval. For a given organ, the incremental probability of death due to radiation-induced cancer is estimated for each year using radiation risk factors and the estimated doses during that year and relevant preceding years. The incremental probabilities of death are used in conjunction with the actuarial life tables to estimate the incremental number of radiation-induced deaths each year for each organ. The number of incremental deaths for the cohort for each organ is obtained by summing the deaths each year over 110 years. The total number of incremental deaths for the cohort is then obtained by summing the total deaths for each organ over all organs.

In addition to providing an estimate of the incremental number of deaths, the life table methodology can be used to estimate the total number of years of life lost to those dying of radiation-induced cancer, the average number of years of life lost per incremental mortality, and the decrease in the population's life expectancy. An example summary table of output from the RADRISK computer code, for inhalation of Ra-226, is shown in Table 4-1.

Risk estimates for chronic irradiation of the cohort may also be applied to a stationary population having the same age-specific mortality rates as the 1970 U.S. population. That is, since the stationary population is formed by superposition of all age groups in the cohort,

TABLE 4-1:
Summary table from RADRISK computer code for Ra-226 inhalation

```
                TOTAL COHORT (1.0E+5 PERSONS) CANCER FATALITIES FROM LIFETIME RA-226   INHALATION
                         AMAD = 1.00, RESP CLEARANCE CLASS =W, F1 = 0.200E+00
                                       FOR 1.0 PCI/YR INTAKE
                                                                                                [12-28-83]

                                                  SUMMARY TABLE

          ADULT    ADULT                  ADULT                 NUMBER OF   AVERAGE    TOTAL     DECREASE     70-YEAR       RISK
          LATENCY  PLATEAU                DEATH      70-YEAR    PREMATURE   YEARS OF   YEARS OF  IN LIFE      DOSE EQUIV-   EQUIVALENT
CANCER    PERIOD   PERIOD   RISK  LET    RATE (DTHS/ DOSE RATE   DEATHS     LIFE LOST  LIFE LOST EXPECTANCY   ALENT RATE    FACTOR
          (YEARS)  (YEARS)               1E6/MRAD/YR) (MRAD/YR)  IN COHORT  (YEARS)    (YEARS)   (YEARS)      (MREM/YR)

LEUKEMIA     2        25    ABS   LOW     2.17E+00   6.83E-05   1.47E-05   2.64E+01   3.88E-04  3.88E-09     2.75E-03      8.39E-04
                                  HIGH    1.74E+01   1.34E-04   2.44E-04   2.72E+01   6.64E-03  6.64E-08
BONE         5        30    ABS   LOW     4.47E-02   9.75E-05   4.48E-07   2.44E+01   1.10E-05  1.10E-10     3.06E-02      2.06E-02
                                  HIGH    8.94E-01   1.52E-03   1.41E-04   2.45E+01   3.46E-03  3.46E-08
THYROID      2        45    ABS   LOW     9.25E-01   2.68E-06   4.14E-07   2.59E+01   1.07E-05  1.07E-10     6.09E-04      2.35E-04
                                  HIGH    7.40E+00   3.03E-05   4.57E-05   2.75E+01   1.26E-03  1.26E-08
BREAST      15       110    ABS   LOW     1.49E+00   3.41E-06   6.16E-07   1.99E+01   1.22E-05  1.22E-10     6.10E-04      2.31E-04
                                  HIGH    1.19E+01   3.03E-05   5.90E-05   2.10E+01   1.24E-03  1.24E-08
LUNG        10       110    ABS   LOW     2.03E+00   7.01E-06   2.53E-06   2.24E+01   5.66E-05  5.66E-10     9.59E-02      3.84E-02
                                  HIGH    1.63E+01   4.80E-03   1.58E-02   2.30E+01   3.64E-01  3.64E-06
STOMACH     15       110    ABS   LOW     9.61E-01   2.14E-06   2.68E-07   2.02E+01   5.42E-06  5.42E-11     6.08E-04      2.30E-04
                                  HIGH    7.69E+00   3.03E-05   3.81E-05   2.10E+01   8.02E-04  8.02E-09
BOWEL       15       110    ABS   LOW     4.47E-01   1.29E-05   9.38E-07   2.12E+01   1.99E-05  1.99E-10     6.95E-04      2.71E-04
                                  HIGH    3.57E+00   3.41E-05   2.01E-05   2.11E+01   4.24E-04  4.24E-09
LIVER       15       110    ABS   LOW     9.77E-01   2.40E-06   2.90E-07   1.99E+01   5.79E-06  5.79E-11     4.28E-04      1.59E-04
                                  HIGH    7.81E+00   2.13E-05   2.67E-05   2.09E+01   5.57E-04  5.57E-09
PANCREAS    15       110    ABS   LOW     6.72E-01   3.33E-06   2.74E-07   1.99E+01   5.44E-06  5.44E-11     6.09E-04      2.23E-04
                                  HIGH    5.16E+00   3.03E-05   2.56E-05   2.10E+01   5.39E-04  5.39E-09
URINARY     15       110    ABS   LOW     2.84E-01   4.63E-06   1.82E-07   2.05E+01   3.74E-06  3.74E-11     7.94E-04      3.05E-04
                                  HIGH    2.27E+00   3.94E-05   1.48E-05   2.11E+01   3.13E-04  3.13E-09
OTHER       15       110    ABS   LOW     1.30E+00   3.33E-06   5.31E-07   1.99E+01   1.06E-05  1.06E-10     6.09E-04      2.32E-04
                                  HIGH    1.04E+01   3.03E-05   5.18E-05   2.10E+01   1.09E-03  1.09E-08

TOTAL (SOMATIC)                                                 1.65E-02   2.31E+01   3.80E-01  3.80E-06     2.08E-02      8.31E-03

30-YEAR GENETIC DOSE COMMITMENTS (MRAD):

   LET       TESTES          OVARIES         AVERAGE
   LOW      5.59E-05        7.44E-05        6.51E-05
   HIGH     8.52E-04        8.52E-04        8.52E-04
```

each age group corresponds to a segment of the stationary population with the total population equal to the sum of all the age groups. Thus, the number of excess fatal cancers calculated for lifetime exposure of the cohort at a constant dose rate would be numerically equal to that calculated for the stationary population receiving a population dose of the same magnitude. This equivalence was used in deriving the risk factors for the environmental pathways analysis described in this report. More detailed discussions of the methodology used by EPA to compute fatal cancer risk estimates are included in Appendices F through H, and in several of the references (Du84, Du80, Su81) for those readers who desire more details than are presented in this section.

In our high-level waste environmental pathway calculations, we have calculated fatal cancer risk commitment factors using the RADRISK data to convert from lifetime radionuclide intake for the inhalation and ingestion pathways and from lifetime external exposure for the air submersion and ground surface pathways directly to fatal cancer risk without calculating dose commitments or dose rates*. As an example, the derivation of an inhalation risk commitment factor will be described in the following paragraphs.

Using the summary tables from the RADRISK computer code output (example in Table 4-1), column 8, total (somatic), gives the total number of premature fatal cancers in a cohort of 100,000 persons born simultaneously and inhaling 1 pCi/y continuously throughout their lifetime. The average lifetime for the persons comprising the cohort is 70.756 years. Thus, the deaths tabulated in column 8 are the result of the collective lifetime intake by the cohort of

$$(1\text{E+5 persons})\left(1\ \frac{\text{pCi}}{\text{yr-person}}\right)(70.756\ \text{yr})\left(1\text{E-12}\ \frac{\text{Ci}}{\text{pCi}}\right) = 7.076\text{E-6 Ci}$$

Then, for a constant lifetime rate of intake, a fatal cancer risk commitment factor can be calculated using the equation

$$\frac{FCF_{np}}{\frac{\text{deaths committed}}{\text{Ci inhaled}}} = \frac{\text{premature deaths in cohort (col. 8, Total Somatic, Table 4-1)}}{7.076\text{E-6 Ci inhaled}} \qquad (4.1\text{-}1)$$

This equation is applicable to a stable population (i.e., a population in which equal numbers of persons are born and die in each year) which has the same age distribution and age specific mortality ratio as used for the cohort. The factors derived using this equation are not applicable for computation of fatal cancer risks to an individual for an acute intake of radioactivity.

For Ra-226 particulates having AMAD = 1.0 micron and respiratory clearance class W, the total number of premature fatal cancers in a cohort of 100,000 persons is 1.65E-2 (Table 4-1, column 8, total (somatic)). Then, applying equation 4.1-1, the fatal cancer risk conversion factor is calculated to be 2,332 $\frac{\text{deaths committed}}{\text{Ci inhaled}}$.

This illustration shows the methodology for deriving the fatal cancer risk conversion factors for inhalation pathways. The procedures used to obtain the factors for the ingestion, air submersion, and ground surface exposure pathways are very similar to those presented above*.

4.2 Analytical Treatment of Daughter Product Buildup During Environmental Transport

Daughter product buildup during environmental transport was not addressed rigorously in the 30 pathway equations discussed in Section 3. An examination of the decay schemes for the nuclides revealed that, in general, the nuclides either (a) had stable daughters, (b) had daughters that were very short-lived compared to the parent, or (c) had moderately to very long-lived first or second daughters. The following techniques were applied as an approximate method of handling daughter product ingrowth during environmental transport.

For case (a), no action to account for daughter product buildup was required.

In case (b), we assumed that the daughter was in secular equilibrium with the parent, i.e., that the activity of the daughter was equal to that of the parent, at all locations of the parent in the environment.

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*Those wishing to compare their fatal cancer risk assessment methodology to that used by EPA should multiply their dosimetry and fatal cancer risk factors for each organ at risk, sum these products, and compare their sums to the EPA risk factors listed in Table 4-2.

Obviously this is a simplifying assumption, since daughter products may behave differently than the parents in the environment. The risk factors for daughters were added to the risk factors for the parent in cases where the daughters were dosimetrically significant. An example of case (b) is Zr-93. Zr-93 has a half life of 1.53E+6yr and an ingestion fatal cancer risk factor of 8.46E-2 fatal cancers per curie intake. The only radioactive daughter of Zr-93 is Nb-93m, which has a half life of 14.6yr and an ingestion fatal cancer risk factor of 4.21E-2. This is clearly a case where Nb-93m is in secular equilibrium with the Zr-93 parent in the environment and we have added the risk factors to obtain the reported risk factor for Zr-93 of 1.27E-1 fatal cancers/Ci ingested (see Appendix B, Table B-1). The rationale is that for each curie of Zr-93 ingested a curie of Nb-93m is present and is also ingested.

In case (c), we performed simple calculations to determine whether a significant buildup of the long-lived daughters would occur during the residence time of the parent in the accessible environment. The accessible environment is that portion of the environment where the material would be available to man. For example, radionuclides present within the root zone of the soil would be in the accessible environment, but radionuclides that have moved below the root zone would have left the accessible environment. Since we believe that the soil root zone is an environmental medium which will have one of the longest retention times for radionuclides in the accessible environment, we calculated the mean lifetime in soil of radionuclides in category (c). The mean lifetime was taken as the reciprocal of the leaching removal rate constant, λ_{Sn}, for the 15 cm soil root zone. Using standard Bateman equations (Ev55), we evaluated the maximum activity of the significant daughter products which could build up during the mean lifetime of the parent and compared this activity to the original activity of the parent. For daughters where the maximum activity exceeded 1 percent of the original parent activity, we added the product of the ingrowth fraction and the daughter risk factor to the parent risk factor. We neglected the contribution of those daughters which built up to less than 1 percent of the original parent activity. Any short-lived daughters in the decay chain between the parent and the first moderate to long-lived daughter are assumed to be in secular equilibrium as described for case (b) above. The specific treatment of daughter products for each parent nuclide in this analysis is specified in Appendix A.

4.3 Application of Risk Factors for Environmental Pathway Calculations

For the inhalation and ingestion pathways two categories of fatal cancer risk factors were calculated which were designated Inhalation 1, Inhalation 2, Ingestion 1 and Ingestion 2. The category 1 factors were used when we believed radionuclides will be present in the accessible environment in a reasonably insoluble chemical form. The category 2 factors were used when we believed the nuclides are in a more soluble chemical form. Class Y risk factors were used for the Inhalation 1 category for all nuclides where they were available and Class W risk factors were used for the Inhalation 2 category where they were available. However, for some radionuclides, only Class D or Class D and Class W inhalation risk factors were available. In these cases, the factors for the least soluble clearance class available were applied. For example, if inhalation risk factors were available for Class D and Class W but not for Class Y, the Class W factors were listed in both the Inhalation 1 and Inhalation 2 categories. For the Ingestion 1 and Ingestion 2 categories, risk factors were used which were derived using the same absorption fraction from gut-to-blood, (f_1), as was used for the Inhalation 1 and Inhalation 2 categories, respectively.

For the volcano/meteorite release mode, the Inhalation 1 and Ingestion 1 risk factors were used and for the other three release modes (releases to rivers, releases to oceans, and releases to land surfaces) the Inhalation 2 and Ingestion 2 risk factors were used. This procedure was followed because we believe that the radionuclides released in the volcano/meteorite release mode will be in a less soluble chemical form than the radionuclides released in the other three release modes.

The risk factors used in our analysis are tabulated in Table 4-2. Included in Appendix B is a tabulation of the risk factors for the parent radionuclides, and the daughter radionuclides where the risk factor was of sufficient magnitude to be added to the risk factors for the parent.

TABLE 4-2:
Fatal Cancer Risk Conversion Factors*

Nuclide	(fatal cancers committed per Ci intake)				$\left(\frac{\text{fatal cancers committed}}{\text{Ci-y/m}^3}\right)$	$\left(\frac{\text{fatal cancers committed}}{\text{Ci-y/m}^2}\right)$
	Inhalation 1	Inhalation 2	Ingestion 1	Ingestion 2	Air Submersion	Ground Contamination
C-14	3.05E-3	3.05E-3	4.32E-1	4.32E-1	0	0
Ni-59	4.76E-1	4.76E-1	3.76E-2	3.76E-2	4.10E-2	8.87E-3
Sr-90	4.52E+2	5.19E+1	2.29E+0	2.85E+1	0	0
Zr-93	2.72E+1	6.60E+0	1.27E-1	1.27E-1	1.23E-1	1.89E-2
Tc-99	6.12E+0	6.12E+0	5.37E-1	5.37E-1	5.97E-4	1.41E-5
Sn-126	5.72E+1	5.72E+1	2.04E+0	2.04E+0	2.54E+3	5.11E+1
I-129	1.61E+1	1.61E+1	2.41E+1	2.41E+1	7.57E+0	3.98E-1
Cs-135	1.27E+0	1.27E+0	1.82E+0	1.82E+0	0	0
Cs-137	8.49E+0	8.49E+0	1.24E+1	1.24E+1	7.18E+2	1.43E+1
Sm-151	5.27E+0	5.27E+0	3.46E-2	3.46E-2	8.15E-4	9.43E-5
Pb-210	2.27E+4	2.99E+3	4.13E+2	4.13E+2	1.34E+0	6.09E-2
Ra-226	4.38E+4	5.33E+3	4.91E+2	4.91E+2	2.35E+3	4.20E+1
Ra-228	7.98E+4	1.58E+4	9.71E+1	9.71E+1	3.43E+3	5.88E+1
Ac-227	6.97E+4	3.74E+4	2.85E+2	2.85E+2	4.81E+2	1.05E+1
Th-229	6.45E+4	2.82E+4	8.55E+1	8.55E+1	3.30E+2	7.46E+0
Th-230	6.89E+4	2.05E+4	5.13E+2	5.13E+2	2.35E+3	4.20E+1

*The fatal cancer risk conversion factors in this table are the sum of the risk factors for the listed nuclide plus any significant daughter products which can grow in during the residence time of the nuclide in the accessible environment. For a more complete discussion, see section 4.2 and Appendices A and B.

TABLE 4-2 Continued:
Fatal Cancer Risk Conversion Factors

Nuclide	(fatal cancers committed per Ci intake)				$\left(\frac{\text{fatal cancers committed}}{\text{Ci-y/m}^3}\right)$	$\left(\frac{\text{fatal cancers committed}}{\text{Ci-y/m}^2}\right)$
	Inhalation 1	Inhalation 2	Ingestion 1	Ingestion 2	Air Submersion	Ground Contamination
Th-232	1.05E+5	2.94E+4	1.17E+2	1.17E+2	3.43E+3	5.88E+1
Pa-231	1.03E+5	6.19E+4	4.67E+2	4.67E+2	5.17E+2	1.14E+1
U-233	2.42E+4	3.70E+3	5.21E+0	5.07E+1	1.68E+1	3.84E-1
U-234	2.07E+4	2.26E+3	9.38E-1	4.61E+1	1.63E-1	1.63E-2
U-235	2.03E+4	2.72E+3	5.86E+0	5.02E+1	2.01+2	4.60E+0
U-236	1.96E+4	2.14E+3	8.86E-1	4.35E+1	1.27E-1	1.48E-2
U-238	1.86E+4	2.04E+3	1.99E+0	4.84E+1	2.36E+1	5.17E-1
Np-237	2.89E+4	2.46E+4	1.86E+2	1.86E+2	2.83E+2	6.39E+0
Pu-238	3.13E+4	2.49E+4	1.86E+2	1.86E+2	8.80E-2	1.68E-2
Pu-239	3.09E+4	2.65E+4	2.04E+1	2.00E+2	9.06E-2	7.64E-3
Pu-240	3.09E+4	2.65E+4	2.04E+1	1.99E+2	8.63E-2	1.61E-2
Pu-241	1.19E+3	1.23E+3	9.57E+0	9.57E+0	5.98E-1	1.87E-2
Pu-242	2.94E+4	2.52E+4	1.94E+1	1.90E+2	7.36E-2	1.34E-2
Am-241	3.27E+4	2.75E+4	2.07E+2	2.07E+2	1.99E+1	6.24E-1
Am-243	3.19E+4	2.73E+4	2.06E+2	2.06E+2	2.55E+2	5.95E+0
Cm-245	6.54E+4	5.58E+4	4.21E+2	4.21E+2	1.02E+2	2.58E+0
Cm-246	3.26E+4	2.78E+4	2.10E+2	2.10E+2	6.81E-2	1.41E-2

4.4 Risk Conversion Factors for Np-237

Neptunium-237 deserves special mention in this document. In ICRP Report 30 (ICRP80) the value for absorption through the gut (f_1) is 10^{-2}. This is a 100-fold increase over the previous value of 10^{-4} recommended in ICRP Report 2 (ICRP60). According to Cohen, this increase in f_1 plus the high cancer risk factor for the liver quoted in BEIR III have caused Np-237 to become a principal nuclide of concern in various assessments of the health effects from disposing of high-level radioactive waste (Co82). In ICRP30, it is mentioned that a value for f_1 of 10^{-3} may be more appropriate for trace quantities of the element or for neptunium incorporated in food. Information published subsequent to the publication of ICRP30 (Th82a, Th82b) suggests that 10^{-3} is probably the most appropriate value for f_1 for neptunium for low environmental levels. The internal dose conversion factors for Np-237 utilized by EPA in this report are based on an f_1 of 10^{-3}; EPA believes, based on current information, that this is the best value to use in these calculations. EPA is aware that ICRP Committee 2 has a task group to recommend the most appropriate values of f_1 for actinides and that the NCRP has established a task group on neptunium. EPA will consider the recommendations, when they become available, of both of these committees in future analyses concerning the disposal of high-level radioactive waste.

Chapter 5: DISCUSSION OF VALUES FOR PARAMETERS*

Some of the parameters used in these calculations appear in several of the 30 pathway equations. For this reason, we decided to discuss parameter values in a separate chapter rather than in the chapters describing the pathway ERC equations. For many parameters, a range of values will be found in the literature. A list of the estimated range of possible values for these parameters is in Appendix C, Section C.9, Table C-10. The parameter values listed in this chapter are used for the fatal cancer risk calculations summarized in Chapter 6 and discussed in more detail in EPA's analysis of the population risks from geologic repositories (EPA85a).

The parameter λ_{Ln} is the rate at which radionuclides are leached from the waste into the groundwater in the repository and is assumed to be the same for all nuclides. A range of values from 1.0 E-2 yr^{-1} to 1.0 E-6 yr^{-1} was examined in the EPA analysis (EPA85a) based on information furnished by the Arthur D. Little Company (Li77b). The value applied in computing fatal cancers per Ci release is 1.0 E-4 yr^{-1}.

The parameter t_{er} is the time after placement in a repository that leaching from the repository begins and usually corresponds to failure of the waste canister. Canister lifetimes ranging between 100 and 5000 years were considered in EPA's analysis of geologic repositories (Sm82, EPA85a). We assume t_{er} is the same for all radionuclides.

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*The variables used in this chapter are defined in the "Nomenclature" section, p. N-1 ff.

The parameter t_{ran} is the time required for radionuclides to travel from the repository to an aquifer. This time varies depending upon the situation being addressed in EPA's analysis of geologic repositories, but the time is usually on the order of a few years.

The parameter t_{arn} is the time required for material to travel from the aquifer to the river. The value chosen for t_{arn} is 760 R_n yr where R_n is the retardation factor for the nuclide being considered. R_n varies between 1 and 10,000 depending on the radionuclide and the situation being considered. Values for R_n are discussed in the A. D. Little report (Li77c) and in EPA's risk assessment report (EPA85a).

The parameter t_{Rn} is the time after placement in a repository that radionuclide n enters the river or ocean and is the sum of t_{er}, t_{ran} and t_{arn}. For these calculations, t_{Rn} is set equal to zero. This is permissible since we are computing the health effects per curie released to the environment. Appropriate non-zero values for t_{er}, t_{ran} and t_{arn} must be selected when computing the total release quantity of radionuclides from a repository.

The parameter Q_{on} is the initial activity of nuclide n in the repository at time t=0. The value for Q_{on} remains constant for various scenarios and is discussed in the risk assessment report (EPA85a).

The parameter λ_{Dn} is the radioactive decay constant for nuclide n. The values for λ_{Dn} are calculated using half-life information from Lederer (Le67) and Kocher (Ko81a) (see Table 5-1).

TABLE 5-1:
Radionuclide decay constants

Nuclide	n	$\lambda_{Dn}(yr^{-1})$
C-14	1	1.21 E-4
Ni-59	2	8.66 E-6
Sr-90	3	2.47 E-2
Zr-93	4	4.62 E-7
Tc-99	5	3.27 E-6
Sn-126	6	6.93 E-6
I-129	7	4.08 E-8
Cs-135	8	2.31 E-7
Cs-137	9	2.31 E-2
Sm-151	10	7.97 E-3
Pb-210	11	3.11 E-2
Ra-226	12	4.33 E-4
Ra-228	13	1.21 E-1
Ac-227	14	3.18 E-2
Th-229	15	9.44 E-5
Th-230	16	9.00 E-6
Th-232	17	4.93 E-11
Pa-231	18	2.12 E-5
U-233	19	4.35 E-6
U-234	20	2.81 E-6
U-235	21	9.85 E-10
U-236	22	2.96 E-8
U-238	23	1.55 E-10
Np-237	24	3.24 E-7
Pu-238	25	8.06 E-3
Pu-239	26	2.84 E-5
Pu-240	27	1.05 E-4
Pu-241	28	4.81 E-2
Pu-242	29	1.83 E-6
Am-241	30	1.51 E-3
Am-243	31	8.72 E-5
Cm-245	32	8.15 E-5
Cm-246	33	1.46 E-4

The parameters f_{LL}, f_{AL}, and f_{AW} are the fractions of the total release for a volcano/meteorite event which are released to the land surface, the air-above-land, and the air-above-water, respectively. The values used for these parameters are $f_{LL} = 0.500$, $f_{AL} = 0.150$, and $f_{AW} = 0.350$.

The parameter P_R is the number of people who drink water from the river and R is the river flow rate. The ratio P_R/R is needed in several equations, and it can be determined for the purpose of this generic evaluation without obtaining site specific data. Utilizing data from Annex D of the 1977 UNSCEAR Report (UN77), the annual flow rate of the rivers of the world is 3 E+16 liters/yr. If one assumes a constant world population of 10^{10} persons,* the ratio P_R/R is 3.3 E-7 person-yr/liter. This value for the ratio P_R/R is within the range of values found for various river basins in the United States. These values ranged from a high for P_R/R of 5.73 E-7 for the Lower Colorado WRC** region to a low of 2.39 E-8 for the Pacific Northwest WRC region based on 1975 river flow and population estimates (see Appendix C, Section C.7).

The parameter I_W is the per capita annual comsumption of drinking water and water-based drinks. The reference adult man drinking rate of water and water-based drinks is 1.65 liters/d (ICRP75), which yields a value of I_w of 603 liters/yr. Note that the fraction of the global flowing water drunk by the world population is approximately 2 E-4.

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*The current world population is about 3.8 E9. However, an estimate of average world population during the time period involved in this calculation is 10^{10} people (UN77).

**WRC - Water Resources Council.

The parameter P_{FF} is the population eating freshwater fish from the river and I_f is the per capita annual freshwater fish consumption rate. The ratio of the population fish consumption rate (P_{FF} I_f) to the river flow rate (R) is needed for the calculation. The UNSCEAR freshwater fish consumption rate for the world is 3.8 E9 man-kg/yr (UN77). We assume that fish consumption will increase proportionately as the population increases from the present level of 3.8 E9 people. For an eventual world population of 10^{10} people, this yields an annual freshwater fish consumption rate of 10^{10} man-kg/yr. Considering the average flow rate of the rivers of the world, the ratio of the population freshwater fish consumption rate to the river flow rate is 10^{10}/3 E16 = 3.3 E-7 man-kg/liter.

The parameter CF_{np} is the bioaccumulation factor for fish or shellfish for nuclide n and pathway p. The primary reference used for CF_{np} for freshwater fish, ocean fish, and ocean shellfish is UCRL-50564, Rev.1 (Th72). The values of the bioaccumulation factors are listed in Table 5-2. The references for the bioacculumation factors not taken from UCRL-50564, Rev. 1 are noted in Table 5-2.

The parameter RI_{np} is the symbol for the terrestrial food pathway factors discussed in Chapter 3. The values used for these factors are listed in Table 5-3. The methodology used to derive these factors is similar to that applied in the AIRDOS-EPA computer code (Mo79) and is

TABLE 5-2:
Bioaccumulation factors for freshwater fish and seafood

Nuclide	n	CF_{np} (Ci/kg per Ci/liter)		
		Freshwater Fish	Ocean Fish	Ocean Shellfish
C-14	1	NA	NA	NA
Ni-59	2	1.00E+2	1.00E+2	2.50E+2
Sr-90	3	1.10E+1 (Ho79)	2.00E+0	2.00E+1
Zr-93	4	3.33E+0	2.00E+2	8.00E+1
Tc-99	5	4.30E+1 (Bl82a)	1.00E+1	5.00E+1
Sn-126	6	3.00E+3	3.00E+3	1.00E+3
I-129	7	3.30E+1 (Ho79)	1.00E+1	5.00E+1
Cs-135	8	1.30E+3 (Ho79)	4.00E+1	2.50E+1
Cs-137	9	1.30E+3 (Ho79)	4.00E+1	2.50E+1
Sm-151	10	2.50E+1	2.50E+1	1.00E+3
Pb-210	11	1.00E+2	3.00E+2	1.00E+3
Ra-226	12	5.00E+1	5.00E+1	1.00E+2
Ra-228	13	5.00E+1	5.00E+1	1.00E+2
Ac-227	14	2.50E+1	2.50E+1	1.00E+3
Th-229	15	3.00E+1	1.00E+3 (Ng84)	2.00E+3
Th-230	16	3.00E+1	1.00E+3 (Ng84)	2.00E+3
Th-232	17	3.00E+1	1.00E+3 (Ng84)	2.00E+3
Pa-231	18	1.10E+1	1.00E+1	1.00E+1
U-233	19	1.00E+1	1.00E+1	1.00E+1
U-234	20	1.00E+1	1.00E+1	1.00E+1
U-235	21	1.00E+1	1.00E+1	1.00E+1
U-236	22	1.00E+1	1.00E+1	1.00E+1
U-238	23	1.00E+1	1.00E+1	1.00E+1
Np-237	24	5.00E+2 (Sc83)	1.00E+2 (Sc83)	1.00E+2 (Sc83)
Pu-238	25	8.00E+0 (Ri83)	3.00E+0	2.00E+2
Pu-239	26	8.00E+0 (Ri83)	3.00E+0	2.00E+2
Pu-240	27	8.00E+0 (Ri83)	3.00E+0	2.00E+2
Pu-241	28	8.00E+0 (Ri83)	3.00E+0	2.00E+2
Pu-242	29	8.00E+0 (Ri83)	3.00E+0	2.00E+2
Am-241	30	8.10E+1 (Ri83)	2.50E+1	1.00E+3
Am-243	31	8.10E+1 (Ri83)	2.50E+1	1.00E+3
Cm-245	32	2.50E+1	2.50E+1	1.00E+3
Cm-246	33	2.50E+1	2.50E+1	1.00E+3

NA - Not Applicable.

TABLE 5-3:
Radionuclide intake factors

Nuclide	RI_{np} (Ci intake per Ci/m^2 deposited) Food Crops	Milk	Meat
C-14	NA	NA	NA
Ni-59	4.38E+0	3.22E-1	2.48E-1
Sr-90	2.57E+0	1.07E+0	8.20E-2
Zr-93	4.21E+0	8.18E-2	2.10E+1
Tc-99	1.57E+0	4.00E+0	1.31E+0
Sn-126	1.10E+0	3.04E-1	9.36E+0
I-129	1.17E+1	1.03E+1	2.78E+0
Cs-135	1.40E+1	8.04E+0	8.84E+0
Cs-137	8.51E-1	1.74E+0	1.91E+0
Sm-151	5.47E-1	4.54E-3	4.37E-1
Pb-210	4.98E-1	5.75E-2	2.66E-2
Ra-226	6.62E-1	1.26E-1	6.26E-2
Ra-228	3.95E-1	9.81E-2	4.53E-2
Ac-227	3.95E-1	4.36E-3	2.10E-3
Th-229	7.33E-1	1.49E-3	6.87E-4
Th-230	2.77E+0	3.87E-3	1.79E-3
Th-232	6.73E+0	8.51E-3	3.93E-3
Pa-231	6.92E-1	1.43E-3	1.10E-3
U-233	1.19E+0	1.57E-1	2.01E-2
U-234	1.19E+0	1.57E-1	2.01E-2
U-235	1.19E+0	1.57E-1	2.01E-2
U-236	1.19E+0	1.57E-1	2.01E-2
U-238	1.19E+0	1.57E-1	2.01E-2
Np-237	5.42E-1	2.52E-3	1.94E-2
Pu-238	3.92E-1	2.17E-5	1.67E-4
Pu-239	4.77E-1	2.37E-5	1.83E-4
Pu-240	4.53E-1	2.32E-5	1.79E-4
Pu-241	3.90E-1	2.17E-5	1.67E-4
Pu-242	4.89E-1	2.40E-5	1.85E-4
Am-241	4.35E-1	9.45E-5	3.18E-4
Am-243	4.87E-1	1.03E-4	3.48E-4
Cm-245	4.10E-1	4.67E-3	3.14E-4
Cm-246	4.08E-1	4.63E-3	3.12E-4

NA - Not Applicable.

discussed, along with input parameter values, more fully in Appendix C. Those readers interested in the details of the derivation of these RI_{np} factors should refer to Appendix C, Section C.1.

The fraction of land used for various food crops is f_p. For the river pathway, irrigation water provides the source of radionuclides to land, so irrigated farmland is the only land of concern. We assume that 50 percent of the irrigated land is used for growing food crops; 25 percent for grazing milk cows; and 25 percent for grazing beef cows. For the non-river release modes, radionuclides reach the land surface via dispersion in air so that the values for the fraction, f_p, must include the portion of U.S. land surface used for farming. It was found that 45 percent of the U.S. land surface was used for farming in 1974 (Wo79). To obtain a value for f_p for the non-river release modes, 45 percent of the value of f_p applied for the river release mode was used. Thus, for the non-river release mode, it was assumed that 23 percent of the land is used for growing food crops; 11 percent for grazing milk cows; and 11 percent for grazing beef cows. The values selected for f_p are upper limits since for all release modes part of the agricultural land will be used for non-food crops (e.g., cotton, etc.).

The fraction of the river flow used for irrigation, f_R, was set equal to 0.1. This number represents the average fraction of total surface water flow used for irrigation for the U.S. and is taken from USGS Circular 765 (Mu77). Data from other publications generally support the choice of 0.1 for a U.S. average (WRC78, WIC70). The value for f_R

varies for various regions of the U.S. from a low of zero for the Ohio and Tennessee regions to a value of almost one for the Lower Colorado region, as shown in Appendix C, Section C.3.

The parameter CP_p is the population that can be fed by raising crop p on a unit area of land. The quantity is obtained by dividing the agricultural productivity (kg/m^2-y) of land on which crops are raised by the annual consumption rate of an individual consuming the crop (kg/y-person). Values for CP_p were obtained from information in two references (Sh82, Ba84) and were derived taking into consideration the types of crops consumed by humans and by milk and beef producing animals. In this analysis, the value of CP_p for food crops is 4.79E-3 persons fed/m^2; for milk, 1.56E-3 persons fed/m^2; and for beef, 7.85E-5 persons fed/m^2. The details of the derivation of these numbers are discussed in Appendix C, Section C.2.

The parameter PD_p is the population density for pathway p. The value used for PD_p is 6.67 E-5 person/m^2, which is the world average population density and was obtained by dividing the assumed world population of 10^{10} persons by the land surface area of the earth of 1.5 E14 m^2 (Wo79). This value is within the range of population densities for various regions of the U.S. (Wo79), as shown in Appendix C, Section C.4.

The parameter RF is the resuspension factor for material resuspending from the ground surface to the air. It is defined as the ratio of air concentration to ground concentration, $X_n(t')/\phi_n(t')$, which is equal to λ_R/v_{gn} where λ_R is a rate constant for resuspension of radionuclides from ground to air and v_{gn} is the deposition velocity from air to land surface. The value of λ_R chosen for this analysis is 10^{-11} sec^{-1}, which should be representative of weathered material (Ne78); and the value used for v_{gn} is 10^{-2} m/sec. The resulting value of RF is 10^{-9} m^{-1} and this value agrees well with the long-term average value suggested by Bennett (Be76). Actually, λ_R and RF are functions of time, but the time dependent relationships were not known and constant values were used to represent the average values over the calculation interval. The value selected for λ_R is appropriate to dry land resuspension, so the dose due to resuspension from wet irrigated land (the river release mode) may be conservatively high.

The parameter I_B is the standard man breathing rate. Based on information contained in ICRP Report 23 (ICRP75), a value of 8400 m^3/yr has been chosen for this parameter.

The parameter SOF is a factor that accounts for the reduction in external dose due to household shielding and occupancy. A conservative value of 1.0 has been chosen for this analysis for all release modes except the river release mode. For the river release mode, the area where external dose could be received is irrigated farm land. After considering the fraction of time a person might spend around irrigated land, we decided to assign a value of 0.33 to SOF for the river release mode.

The rate constant for transfer of nuclides from available to unavailable soil is λ_{Sn}. The value for λ_{Sn} is nuclide dependent and is determined using a method (Ba79a) and data (Ba84) described by Baes. The values used for λ_{Sn} in this analysis are given in Table 5-4. Baes models the loss of radionuclides from the soil root zone due to water

TABLE 5-4:
Leaching coefficients for radionuclides in soil

Element	Leaching Coefficient (λ_{Sn}) (yr^{-1})
C	NA
Ni	5.40E-3
Sr	2.31E-2
Zr	2.70E-4
Tc	4.90E-1
Sn	3.24E-3
I	1.57E-3
Cs	8.10E-4
Sm (Rare Earth)	1.25E-3
Pb	9.00E-4
Ra	1.80E-3
Ac	5.40E-4
Th	5.40E-6
Pa	3.24E-4
U	1.80E-3
Np	2.69E-2
Pu	1.80E-4
Am	1.16E-3
Cm	4.05E-4

NA - Not Applicable.

leaching but does not consider erosion of soil (and radionuclides) as a removal mechanism. For long time periods, erosion may be a significant removal mechanism which would make the Baes model conservative for this application.

The risk conversion factors used for radionuclides deposited to the ground were derived using the assumption that the material remains on the surface. Over the long time periods involved in these calculations, the radionuclides will move vertically downward into the soil. The gamma radiation emitted from these nuclides is partially shielded by the soil such that the dose equivalent per unit deposition is less than given by the surface dose conversion factors. We derived correction factors for each nuclide by assuming that the radioactivity was uniformly distributed vertically within the soil root zone (see the discussion in Section 3.1.6). These correction factors are listed in Table 5-5 and were derived using the methodology discussed in Appendix C, Section C.5.

The parameter f_{wt} is the fraction of a radionuclide which passes through a drinking water treatment plant and remains in potable water. We conservatively assumed a value of 1.0 for all radionuclides. However, values in the literature will range from 0.2 to 1.0 for the radionuclides considered in this analysis (F171, De75, NRC78).

TABLE 5-5:
Ground surface radionuclide correction factors (GC_{np})

Nuclide	GC_{np} (Dimensionless)
C-14	Beta emitter
Ni-59	8.8E-5
Sr-90	Beta emitter
Zr-93	1.3E-3
Tc-99	Beta emitter
Sn-126	2.2E-1
I-129	1.0E-2
Cs-135	Beta emitter
Cs-137	2.4E-1
Sm-151	Beta emitter
Pb-210	2.3E-2
Ra-226	2.4E-1
Ra-228	1.8E-1
Ac-227	1.5E-1
Th-229	9.7E-2
Th-230	2.4E-1
Th-232	1.8E-1
Pa-231	1.3E-1
U-233	7.5E-2
U-234	2.9E-2
U-235	7.3E-2
U-236	2.5E-4
U-238	4.3E-2
Np-237	9.2E-2
Pu-238	3.8E-4
Pu-239	4.1E-4
Pu-240	4.0E-4
Pu-241	1.2E-2
Pu-242	4.0E-4
Am-241	1.2E-2
Am-243	6.9E-2
Cm-245	3.3E-2
Cm-246	4.0E-4

The parameter f_{sw} is the fraction of drinking water which is supplied by surface water. State average values varied from a high of 0.90 for Maryland to a low of 0.08 for Idaho with the U.S. average being ~ 0.65 (Mu77). We used the U.S. average value in these calculations.

The transfer rate coefficients used in the ocean model are γ_1 and γ_2. That for movement of water from the lower to the upper ocean compartment is γ_2 and the value of it used for this analysis is 6.25 E-4 yr^{-1}(Ma73). For movement of water from the upper to the lower ocean compartment, the transfer rate coefficient is $^{\gamma}1$, and the value applied for this analysis is 3.3 E-2 yr^{-1}, which is derived using the methodology discussed in Appendix C, Section C.10.

The parameters SF_{1n} and SF_{2n} are the sedimentation coefficients from the upper layer and the lower layer of the ocean, respectively, for nuclide n. The treatment of sedimentation is taken from a report of the working group of the International Nuclear Fuel Cycle Evaluation (INFCE78), and the methodology used to derive nuclide specific values for SF_{1n} and SF_{2n} is discussed in Appendix C, Section C.10. The values we used for these two parameters are listed in Table 5-6.

The ratio of the population seafood consumption rate (P_p I_p) to the ocean upper compartment volume (V_1) is needed in these calculations. P_p is the population eating seafood and I_p is the per

TABLE 5-6:
Values for ocean sedimentation coefficients (SF_{1n} and SF_{2n})

Element	Sedimentation Coefficients (yr^{-1}) SF_{1n}	SF_{2n}
C	0	0
Ni	5.33E-6	1.02E-7
Sr	2.67E-7	5.10E-9
Zr	2.67E-4	5.10E-6
Tc	0	0
Sn	0	0
I	0	0
Cs	2.67E-6	5.10E-8
Sm (Rare Earth)	4.00E-5	7.64E-7
Pb	2.67E-4	5.10E-6
Ra	1.33E-6	2.55E-8
Ac	6.67E-5	1.27E-6
Th	2.00E-3	3.82E-5
Pa	2.67E-4	5.10E-6
U	4.00E-5	7.64E-7
Np	1.33E-7	2.55E-9
Pu	2.67E-4	5.10E-6
Am	1.33E-4	2.55E-6
Cm	4.00E-5	7.64E-7

capita seafood annual consumpion rate for pathway p (p = 9 for fish ingestion and p = 10 for shellfish ingestion). V_1 is the volume of the upper compartment of the ocean where it is assumed that the edible fish are harvested. For this generic analysis, average values for the world are used. For ocean fish, the projected average population consumption rate is 6 kg/yr$\cdot 10^{10}$ persons = 6 E10 kg-persons/yr and for ocean shellfish it is 1 kg/yr$\cdot 10^{10}$ persons = 1 E10 kg-persons/yr (UN77). The volume of the upper compartment of the ocean is obtained by multiplying the world ocean surface area of 3.6 E14 m^2 (CRC75) by the assumed depth of the upper compartment of the ocean of 75 m. The resulting volume is 2.7 E16 m^3 or 2.7 E19 liters. Thus the ratio $P_p \cdot I_p/V_1$ is 2.2 E-9 kg-people/liter-yr for ocean fish and 3.7 E-10 kg-people/liter-yr for ocean shellfish.

The terms t_L and t_V represent the times after placement in a repository that radioactive material is transmitted to the land surface or into air for the land surface and the volcano/meteorite pathways, respectively. The values for t_L and t_V change with various scenarios and discussion of the values chosen for these parameters for various scenarios is included in the risk assessment report (EPA85a). However, for this analysis, where we are calculating the fatal cancers per Ci released to the available environment, we have set t_L and t_V equal to zero.

The average height of the troposphere is h_A. Tropospheric height ranges from 25000 ft (7600 m) to 60,000 ft (18,000 m) (Wo79). An average value of 13,000 m was selected for these calculations.

The deposition velocity from air to oceans is v_{wn}. In this analysis, a value of 2 cm/sec was used for all nuclides, which was derived from general deposition velocity information contained in Meteorology and Atomic Energy, 1968 (AEC68).

The rate constant for deposition from air to ocean is λ_{wn}, which is equal to v_{wn}/h_A. Thus, the value calculated for this analysis is 48.7 yr^{-1}.

Chapter 6: HEALTH EFFECTS PER CURIE RELEASE RESULTS*

6.1 Fatal Cancers per Curie Release to the Accessible Environment

In this section, we list the fatal cancers in the first 10,000 years per curie release to the accessible environment for all nuclides considered in the EPA environmental transport pathway analysis. These fatal cancer estimates have been computed using the methodology and algorithms presented in Chapters 2 and 3 and the parameter values discussed and listed in Chapters 4 and 5. These fatal cancer estimates have been used to develop the radionuclide release limits in Table 1 of EPA's environmental standards for high-level and transuranic radioactive waste disposal 40CFR191 (EPA85c). Fatal cancer estimates for each of the four release modes, for each radionuclide, are shown in Table 6-1. Tables 6-2, 6-3, and 6-4 show the contributions of the various environmental pathways to the fatal cancers per curie values for the river, ocean, and land surface release modes. As mentioned previously, a period of 10,000 years was used for the integration time. A value of 10^{-4} parts per year was used for the waste leach rate (λ_{Ln}), although these fatal cancers per curie parameters have only a very slight dependence on the value chosen for λ_{Ln}.

For the fatal cancer estimates listed in Tables 6-1 through 6-5, the dose response functions applied are linear for both low and high-LET radiation. For low-LET radiation, EPA considers plausible two of the dose response functions discussed in the BEIR-III report (NAS80). They are the linear model and the linear-quadratic model; with the linear model being more conservative. In Appendix G, where we have discussed the methodology employed in estimating the risk of fatal cancers resulting from radionuclide releases, we have presented information on both the linear and the linear-quadratic model for low-LET radiation so that the reader can compare the magnitude of the risk estimates obtained for given doses using both models. The use of the linear model for both low and high-LET radiation in our calculation has been thoroughly reviewed and accepted by the High-Level Radioactive Waste Disposal Subcommittee of the EPA Science Advisory Board (EPA84a).

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*The variables used in this chapter are defined in the "Nomenclature" section, p. N-1 ff.

TABLE 6-1:
Fatal cancers per curie released to the accessible environment for different release modes

Nuclide	Releases to a River	Releases to an Ocean	Releases to Land Surface	Releases due to Violent Interactions*
C - 14	5.83E-02	5.83E-02	5.83E-02	5.83E-02
Ni- 59	4.61E-05	1.70E-06	6.79E-07	2.89E-05
Sr- 90	2.25E-02	6.14E-06	3.76E-05	1.16E-03
Zr- 93	1.51E-04	8.59E-06	2.26E-05	1.22E-04
Tc- 99	3.65E-04	3.17E-06	5.65E-08	1.99E-04
Sn-126	1.05E-02	2.07E-03	1.38E-03	2.73E-02
I -129	8.07E-02	1.43E-04	3.96E-03	5.57E-02
Cs-135	7.73E-03	2.61E-05	5.75E-04	4.91E-03
Cs-137	1.07E-02	2.28E-05	2.19E-05	3.39E-03
Sm-151	9.38E-06	4.01E-07	6.71E-08	4.72E-06
Pb-210	1.18E-01	6.80E-03	1.52E-04	4.31E-02
Ra-226	1.63E-01	5.40E-03	5.62E-03	7.20E-02
Ra-228	2.41E-02	1.07E-04	1.57E-05	2.78E-02
Ac-227	6.67E-02	1.94E-03	1.24E-04	3.82E-02
Th-229	3.49E-02	2.71E-02	1.90E-02	5.06E-02
Th-230	5.38E-01	1.87E-01	3.86E-01	1.26E+00
Th-232	3.40E-01	4.32E-02	3.76E-01	3.73E-01
Pa-231	1.48E-01	1.67E-03	2.36E-02	1.28E-01
U -233	2.15E-02	1.90E-04	7.51E-04	7.75E-03
U -234	1.96E-02	1.73E-04	6.54E-04	5.94E-03
U -235	2.17E-02	1.89E-04	8.42E-04	8.27E-03
U -236	1.85E-02	1.64E-04	6.18E-04	5.62E-03
U -238	2.06E-02	1.83E-04	6.90E-04	5.67E-03
Np-237	7.95E-02	7.03E-03	1.21E-04	2.83E-02
Pu-238	4.23E-02	4.06E-04	3.10E-04	2.07E-02
Pu-239	4.97E-02	2.19E-03	6.23E-03	1.20E-02
Pu-240	4.84E-02	1.91E-03	5.22E-03	1.15E-02
Pu-241	2.17E-03	9.24E-06	2.50E-06	9.36E-04
Pu-242	4.79E-02	2.18E-03	6.34E-03	1.09E-02
Am-241	5.42E-02	3.74E-03	1.05E-03	2.54E-02
Am-243	5.72E-02	1.09E-02	2.45E-03	3.40E-02
Cm-245	1.01E-01	2.29E-02	8.08E-03	6.09E-02
Cm-246	4.99E-02	1.01E-02	3.54E-03	2.89E-02

*For example, interactions of a metorite or a volcanic eruption with a repository.

TABLE 6-2:
Fatal cancers per curie released to the accessible environment for releases to a river

Nuclide	TOTAL	Drinking Water Ingestion (p = 1)	Freshwater Fish Ingestion (p = 2)	Above Surface Crops Ingestion (p = 3)	Milk Ingestion (p = 4)	Beef Ingestion (p = 5)	Inhalation of Resuspended Material (p = 6)	External Dose - Ground Contam. (p = 7)	External Dose - Air Submersion (p = 8)
C - 14	5.83E-02	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Ni- 59	4.61E-05	4.91E-06	1.25E-06	3.94E-05	4.72E-07	1.83E-08	3.25E-10	3.17E-10	1.11E-15
Sr- 90	2.25E-02	3.72E-03	1.04E-04	1.75E-02	1.19E-03	4.59E-06	4.05E-09	0.00E+00	0.00E+00
Zr- 93	1.51E-04	1.66E-05	1.41E-07	1.28E-04	4.05E-07	5.23E-06	6.58E-08	1.45E-07	4.86E-14
Tc- 99	3.65E-04	7.02E-05	7.70E-06	2.02E-04	8.38E-05	1.38E-06	4.67E-11	0.00E+00	1.80E-19
Sn-126	1.05E-02	2.67E-04	2.04E-03	5.37E-04	2.42E-05	3.75E-05	6.47E-08	7.55E-03	1.14E-10
I -129	8.07E-02	3.15E-03	2.65E-04	6.75E-02	9.68E-03	1.31E-04	3.68E-08	5.41E-06	6.86E-13
Cs-135	7.73E-03	2.38E-04	7.89E-04	6.10E-03	5.71E-04	3.16E-05	5.38E-09	0.00E+00	0.00E+00
Cs-137	1.07E-02	1.62E-03	5.37E-03	2.53E-03	8.42E-04	4.65E-05	1.33E-09	3.19E-04	4.45E-12
Sm-151	9.38E-06	4.52E-06	2.88E-07	4.53E-06	6.13E-09	2.97E-08	2.14E-09	0.00E+00	1.31E-17
Pb-210	1.18E-01	5.40E-02	1.38E-02	4.93E-02	9.26E-04	2.16E-05	3.45E-07	9.60E-08	6.13E-15
Ra-226	1.63E-01	6.41E-02	8.18E-03	7.78E-02	2.41E-03	6.03E-05	8.91E-06	1.00E-02	1.56E-10
Ra-228	2.41E-02	1.27E-02	1.62E-03	9.19E-03	3.71E-04	8.63E-06	5.61E-07	2.23E-04	4.83E-12
Ac-227	6.67E-02	3.72E-02	2.37E-03	2.70E-02	4.85E-05	1.17E-06	4.29E-06	1.07E-04	2.18E-12
Th-229	3.49E-02	1.12E-02	8.55E-04	1.50E-02	4.97E-06	1.15E-07	4.85E-04	7.39E-03	2.25E-10
Th-230	5.38E-01	6.70E-02	5.13E-03	3.40E-01	7.74E-05	1.80E-06	4.29E-04	1.25E-01	1.95E-09
Th-232	3.40E-01	1.53E-02	1.17E-03	1.89E-01	3.88E-05	9.02E-07	6.27E-04	1.34E-01	2.90E-09
Pa-231	1.48E-01	6.10E-02	1.71E-03	7.74E-02	2.60E-05	1.01E-06	5.33E-04	7.58E-03	1.76E-10
U -233	2.15E-02	6.62E-03	1.69E-04	1.44E-02	3.10E-04	2.00E-06	7.41E-06	3.43E-05	1.33E-12
U -234	1.96E-02	6.02E-03	1.54E-04	1.31E-02	2.82E-04	1.82E-06	4.53E-06	5.63E-07	1.29E-14
U -235	2.17E-02	6.56E-03	1.67E-04	1.43E-02	3.07E-04	1.98E-06	5.46E-06	4.00E-04	1.60E-11
U -236	1.85E-02	5.68E-03	1.45E-04	1.24E-02	2.66E-04	1.72E-06	4.29E-06	4.41E-09	1.01E-14
U -238	2.06E-02	6.32E-03	1.61E-04	1.38E-02	2.96E-04	1.91E-06	4.09E-06	2.65E-05	1.88E-12
Np-237	7.95E-02	2.43E-02	3.10E-02	2.41E-02	1.83E-05	7.08E-06	3.40E-06	4.83E-05	1.55E-12
Pu-238	4.23E-02	2.43E-02	4.96E-04	1.75E-02	1.57E-07	6.10E-08	1.14E-05	1.74E-09	1.60E-15
Pu-239	4.97E-02	2.61E-02	5.33E-04	2.28E-02	1.85E-07	7.18E-08	3.14E-04	2.21E-08	4.26E-14
Pu-240	4.84E-02	2.60E-02	5.31E-04	2.16E-02	1.80E-07	6.99E-08	2.75E-04	3.97E-08	3.55E-14
Pu-241	2.17E-03	1.25E-03	2.55E-05	8.94E-04	8.10E-09	3.14E-09	8.73E-08	9.46E-09	1.68E-15
Pu-242	4.79E-02	2.48E-02	5.07E-04	2.23E-02	1.78E-07	6.90E-08	3.13E-04	3.95E-08	3.62E-14
Am-241	5.42E-02	2.70E-02	5.59E-03	2.16E-02	7.63E-07	1.29E-07	3.85E-05	6.22E-06	1.10E-12
Am-243	5.72E-02	2.69E-02	5.56E-03	2.40E-02	8.28E-07	1.41E-07	7.92E-05	7.08E-04	2.93E-11
Cm-245	1.01E-01	5.50E-02	3.51E-03	4.13E-02	7.67E-05	2.59E-07	3.85E-04	3.49E-04	2.79E-11
Cm-246	4.99E-02	2.74E-02	1.75E-03	2.05E-02	3.79E-05	1.29E-07	1.75E-04	2.11E-08	1.70E-14

TABLE 6-3:
Fatal cancers per curie released to the accessible environment for releases to an ocean

Nuclide	TOTAL	Ocean Fish Ingestion (p = 9)	Ocean Shellfish Ingestion (p = 10)
C - 14	5.83E-02	N/A	N/A
Ni- 59	1.70E-06	1.20E-06	5.00E-07
Sr- 90	6.14E-06	2.30E-06	3.84E-06
Zr- 93	8.59E-06	8.05E-06	5.37E-07
Tc- 99	3.17E-06	1.73E-06	1.44E-06
Sn-126	2.07E-03	1.96E-03	1.09E-04
I -129	1.43E-04	7.82E-05	6.51E-05
Cs-135	2.61E-05	2.36E-05	2.46E-06
Cs-137	2.28E-05	2.07E-05	2.15E-06
Sm-151	4.01E-07	5.23E-08	3.49E-07
Pb-210	6.80E-03	4.42E-03	2.46E-03
Ra-226	5.40E-03	4.05E-03	1.35E-03
Ra-228	1.07E-04	8.02E-05	2.67E-05
Ac-227	1.94E-03	2.53E-04	1.69E-03
Th-229	2.71E-02	2.03E-02	6.76E-03
Th-230	1.87E-01	1.40E-01	4.67E-02
Th-232	4.32E-02	3.24E-02	1.08E-02
Pa-231	1.67E-03	1.43E-03	2.38E-04
U -233	1.90E-04	1.63E-04	2.71E-05
U -234	1.73E-04	1.48E-04	2.47E-05
U -235	1.89E-04	1.62E-04	2.70E-05
U -236	1.64E-04	1.41E-04	2.34E-05
U -238	1.83E-04	1.57E-04	2.61E-05
Np-237	7.03E-03	6.03E-03	1.00E-03
Pu-238	4.06E-04	3.35E-05	3.73E-04
Pu-239	2.19E-03	1.81E-04	2.01E-03
Pu-240	1.91E-03	1.57E-04	1.75E-03
Pu-241	9.24E-06	7.63E-07	8.48E-06
Pu-242	2.18E-03	1.80E-04	2.00E-03
Am-241	3.74E-03	4.88E-04	3.25E-03
Am-243	1.09E-02	1.42E-03	9.44E-03
Cm-245	2.29E-02	2.98E-03	1.99E-02
Cm-246	1.01E-02	1.31E-03	8.75E-03

TABLE 6-4:
Fatal cancers per curie released to the accessible environment for releases to a land surface

Nuclide	TOTAL	Above Surface Crops Ingestion (p = 13)	Milk Ingestion (p = 14)	Beef Ingestion (p = 15)	Inhalation of Resuspended Material (p = 12)	External Dose - Ground Contam. (p = 16)	External Dose - Air Submersion (p = 11)
C - 14	5.83E-02	N/A	N/A	N/A	N/A	N/A	N/A
Ni- 59	6.79E-07	6.68E-07	7.65E-09	2.96E-10	3.11E-09	3.54E-11	3.19E-14
Sr- 90	3.76E-05	3.53E-05	2.29E-06	8.83E-09	4.02E-08	0.00E+00	0.00E+00
Zr- 93	2.26E-05	2.11E-05	6.39E-08	8.25E-07	4.47E-07	1.91E-07	9.92E-13
Tc- 99	5.65E-08	3.99E-08	1.58E-08	2.61E-10	4.66E-10	0.00E+00	5.41E-18
Sn-126	1.38E-03	1.46E-05	6.30E-07	9.76E-07	6.03E-07	1.37E-03	3.19E-09
I -129	3.96E-03	3.47E-03	4.76E-04	6.47E-06	3.23E-07	1.89E-06	1.81E-11
Cs-135	5.75E-04	5.25E-04	4.70E-05	2.60E-06	4.32E-08	0.00E+00	0.00E+00
Cs-137	2.19E-05	1.01E-05	3.22E-06	1.78E-07	1.31E-08	8.33E-06	1.32E-10
Sm-151	6.71E-08	4.61E-08	5.96E-11	2.89E-10	2.07E-08	0.00E+00	3.81E-16
Pb-210	1.52E-04	1.46E-04	2.63E-06	6.11E-08	3.41E-06	1.86E-09	1.82E-13
Ra-226	5.62E-03	2.96E-03	8.78E-05	2.19E-06	7.88E-05	2.49E-03	4.13E-09
Ra-228	1.57E-05	8.44E-06	3.26E-07	7.58E-09	5.59E-06	1.34E-06	1.44E-10
Ac-227	1.24E-04	7.94E-05	1.36E-07	3.31E-09	4.24E-05	2.06E-06	6.49E-11
Th-229	1.90E-02	3.44E-03	1.09E-06	2.53E-08	2.77E-03	1.27E-02	3.85E-09
Th-230	3.86E-01	9.63E-02	2.10E-05	4.88E-07	2.56E-03	2.87E-01	3.49E-08
Th-232	3.76E-01	5.46E-02	1.08E-05	2.50E-07	3.76E-03	3.18E-01	5.23E-08
Pa-231	2.36E-02	1.13E-02	3.65E-06	1.41E-07	3.69E-03	8.53E-03	3.66E-09
U -233	7.51E-04	6.61E-04	1.36E-05	8.75E-08	6.59E-05	1.06E-05	3.56E-11
U -234	6.54E-04	6.01E-04	1.24E-05	7.96E-08	4.03E-05	1.74E-07	3.46E-13
U -235	8.42E-04	6.56E-04	1.35E-05	8.68E-08	4.85E-05	1.24E-04	4.27E-10
U -236	6.18E-04	5.68E-04	1.17E-05	7.52E-08	3.82E-05	1.37E-09	2.70E-13
U -238	6.90E-04	6.32E-04	1.30E-05	8.37E-08	3.64E-05	8.21E-06	5.01E-11
Np-237	1.21E-04	8.57E-05	6.21E-08	2.40E-08	3.36E-05	1.12E-06	4.61E-11
Pu-238	3.10E-04	2.00E-04	1.73E-09	6.68E-10	1.10E-04	1.30E-10	4.63E-14
Pu-239	6.23E-03	4.20E-03	3.25E-08	1.26E-08	2.03E-03	3.21E-08	8.25E-13
Pu-240	5.22E-03	3.47E-03	2.77E-08	1.08E-08	1.75E-03	4.71E-08	6.78E-13
Pu-241	2.50E-06	1.64E-06	1.42E-11	5.49E-12	8.66E-07	1.13E-10	5.01E-14
Pu-242	6.34E-03	4.30E-03	3.29E-08	1.28E-08	2.04E-03	6.23E-08	7.09E-13
Am-241	1.05E-03	7.00E-04	2.37E-08	4.01E-09	3.46E-04	1.32E-06	2.98E-11
Am-243	2.45E-03	1.49E-03	4.91E-08	8.35E-09	6.63E-04	2.96E-04	7.37E-10
Cm-245	8.08E-03	5.04E-03	8.93E-06	3.02E-08	2.73E-03	3.07E-04	5.94E-10
Cm-246	3.54E-03	2.29E-03	4.06E-06	1.38E-08	1.24E-03	1.64E-08	3.62E-13

6.2 Comparison of Fatal Cancers and Serious Genetic Effects (All Generations) per Curie Release to the Accessible Environment

The curie release limits for 40CFR191 are based on fatal cancer risks. The estimated fatal cancers per curie of various radionuclides released to the accessible environment are summarized, for the four release modes we considered, in Table 6-1.

We also computed estimates of the serious genetic effects to all future generations per curie release to the accessible environment. The mechanics of computing the estimates of serious genetic effects is the same as is described in this report for fatal cancer risks except that genetic effects risk factors are applied in place of the fatal cancer risk factors (FCF_{np}). The genetic effects risk factors are derived using the genetic risk estimates from BEIR-3 (NAS80) and using the dose received before age 30. The BEIR-3 estimates are "indirect" estimates and are calculated using the normal prevalence of genetic defects and the dose that is considered to double this risk. The NAS estimates used by EPA are based on a doubling dose range, with a lower bound of 50 rem and an upper bound of 250 rem. To express the range as a single estimate, the geometric mean of the range is used, a method first recommended by UNSCEAR (UN58) for purposes of calculating genetic risk. A factor of three increase in risk for high dose rate, low-LET radiation is also used. We apply an RBE of 20 to estimate the genetic risks for all high-LET radiations.

In developing the average mutation rate for the two sexes used in the calculation of the relative mutation risk, the BEIR-3 committee postulated that the induced mutation rate in females was about 40 percent of that in males (NAS80). Recent studies by Dobson et al. (Do83a, Do83b, Do84a, Do84b) suggest that the assumption was invalid and that human oocytes should have a risk equivalent to that of human spermatogonia. Use of the results of the Dobson studies in place of the BEIR-3 postulation would increase our genetic risk estimates by a factor of 1.43.

A discussion of radiation-induced health effects other than fatal cancer is included in section G.5 of Appendix G. Specifically a more detailed discussion of the basis for the EPA genetic risk estimates is incorporated in section G.5.5.

In Table 6-5, we have listed our estimates for both the fatal cancers and the serious genetic effects to all generations so that the reader can compare the relative magnitude of these two health effects. In reviewing Table 6-5, we note that the genetic effects are about 50 percent of the fatal cancers for ^{126}Sn, ^{135}Cs, ^{137}Cs, ^{228}Ra, and ^{232}Th and 25 to 50 percent for ^{59}Ni, ^{226}Ra, ^{227}Ac, ^{229}Th, ^{230}Th, and ^{231}Pa. The genetic effects are between 15 and 25 percent of the fatal cancers for ^{210}Pb, ^{237}Np and all Pu, Am, and Cm isotopes. For the other radionuclides, the genetic effects are less than 15 percent of the fatal cancer estimates. Our curie release limits for 40 CFR 191 were based only on consideration of fatal cancers because we believe that the consideration of genetic and other effects, along with the fatal cancers, would not significantly effect the results.

TABLE 6-5:
Comparison of fatal cancers and genetic effects per curie release to the accessible environment for different release modes

Nuclide	Release to a River		Release to an Ocean		Release to Land Surface		Releases due to Violent Interactions*	
	Fatal Cancers	Genetic	Fatal Cancers	Genetic	Fatal Cancers	Genetic	Fatal Cancers	Genetic
C - 14	5.83E-02	3.03E-02	5.83E-02	3.03E-02	5.83E-02	3.03E-02	5.83E-02	3.03E-02
Ni- 59	4.61E-05	1.55E-05	1.70E-06	5.74E-07	6.79E-07	2.29E-07	2.89E-05	9.73E-06
Sr- 90	2.25E-02	4.40E-04	6.14E-06	1.20E-07	3.76E-05	7.34E-07	1.16E-03	8.40E-06
Zr- 93	1.51E-04	9.36E-06	8.59E-06	5.31E-07	2.26E-05	1.44E-06	1.22E-04	7.23E-06
Tc- 99	3.65E-04	1.74E-05	3.17E-06	1.51E-07	5.65E-08	2.66E-09	1.99E-04	9.37E-06
Sn-126	1.05E-02	4.22E-03	2.07E-03	6.57E-04	1.38E-03	6.01E-04	2.73E-02	1.17E-02
I -129	8.07E-02	1.82E-04	1.43E-04	3.19E-07	3.96E-03	9.79E-06	5.57E-02	1.34E-04
Cs-135	7.73E-03	3.19E-03	2.61E-05	1.07E-05	5.75E-04	2.37E-04	4.91E-03	2.02E-03
Cs-137	1.07E-02	4.13E-03	2.28E-05	8.77E-06	2.19E-05	8.83E-06	3.39E-03	1.36E-03
Sm-151	9.38E-06	1.24E-09	4.01E-07	5.28E-11	6.71E-08	6.45E-12	4.72E-06	4.59E-10
Pb-210	1.18E-01	1.91E-02	6.80E-03	1.11E-03	1.52E-04	2.42E-05	4.31E-02	6.01E-03
Ra-226	1.63E-01	3.54E-02	5.40E-03	1.10E-03	5.62E-03	1.69E-03	7.20E-02	1.26E-02
Ra-228	2.41E-02	1.11E-02	1.07E-04	4.91E-05	1.57E-05	4.70E-06	2.78E-02	3.08E-03
Ac-227	6.67E-02	1.57E-02	1.94E-03	4.53E-04	1.24E-04	2.61E-05	3.82E-02	5.13E-03
Th-229	3.49E-02	8.31E-03	2.71E-02	5.09E-03	1.90E-02	6.13E-03	5.06E-02	7.26E-03
Th-230	5.38E-01	1.35E-01	1.87E-01	3.64E-02	3.86E-01	1.42E-01	1.26E+00	4.32E-01
Th-232	3.40E-01	1.36E-01	4.32E-02	1.65E-02	3.76E-01	1.57E-01	3.73E-01	1.37E-01
Pa-231	1.48E-01	2.33E-02	1.67E-03	2.38E-04	2.36E-02	5.57E-03	1.28E-01	2.38E-02
U -233	2.15E-02	2.03E-03	1.90E-04	1.77E-05	7.51E-04	6.79E-05	7.75E-03	1.81E-04
U -234	1.96E-02	1.66E-03	1.73E-04	1.46E-05	6.54E-04	5.19E-05	5.94E-03	9.10E-06
U -235	2.17E-02	2.06E-03	1.89E-04	1.68E-05	8.42E-04	1.11E-04	8.27E-03	7.47E-04
U -236	1.85E-02	1.57E-03	1.64E-04	1.39E-05	6.18E-04	4.91E-05	5.62E-03	8.08E-06
U -238	2.06E-02	1.50E-03	1.83E-04	1.32E-05	6.90E-04	5.02E-05	5.67E-03	4.98E-05
Np-237	7.95E-02	1.29E-02	7.03E-03	1.14E-03	1.21E-04	1.93E-05	2.83E-02	3.63E-03
Pu-238	4.23E-02	7.23E-03	4.06E-04	6.94E-05	3.10E-04	5.12E-05	2.07E-02	2.42E-03
Pu-239	4.97E-02	8.05E-03	2.19E-03	3.54E-04	6.23E-03	9.76E-04	1.20E-02	7.08E-04
Pu-240	4.84E-02	7.85E-03	1.91E-03	3.10E-04	5.22E-03	8.21E-04	1.15E-02	6.73E-04
Pu-241	2.17E-03	3.20E-04	9.24E-06	1.36E-06	2.50E-06	3.61E-07	9.36E-04	1.06E-04
Pu-242	4.79E-02	7.71E-03	2.18E-03	3.52E-04	6.34E-03	9.92E-04	1.09E-02	6.50E-04
Am-241	5.42E-02	8.89E-03	3.74E-03	6.13E-04	1.05E-03	1.67E-04	2.54E-02	3.06E-03
Am-243	5.72E-02	9.45E-03	1.09E-02	1.75E-03	2.45E-03	4.62E-04	3.40E-02	5.04E-03
Cm-245	1.01E-01	1.64E-02	2.29E-02	3.69E-03	8.08E-03	1.35E-03	6.09E-02	7.56E-03
Cm-246	4.99E-02	8.08E-03	1.01E-02	1.63E-03	3.54E-03	5.55E-04	2.89E-02	3.54E-03

*For example, interactions of a metorite or a volcanic eruption with a repository.

Chapter 7: UNCERTAINTY ANALYSIS*

Environmental pathway dosimetry and risk models generally employ environmental transport methodology consisting of multiplicative chain algorithms incorporating several variables. In performing regulatory analyses, there is a tendency to choose conservative values for these variables due to the inherent uncertainty in the parameters. The multiplicative nature of the models means that conservatisms in chosen values for individual parameters can lead to larger conservatism in the result. This becomes an issue of contention between the regulators and those being regulated in virtually every rulemaking action where regulatory decisions will be made based on the results of the application of multiplicative chain models. Since the protection of public health is at stake, regulators believe that they must choose conservative values to "insure" that the public is protected. The problem with this approach is that multiple conservatisms can lead to extremely conservative and sometimes unrealistic results. Those being regulated generally experience escalating project costs as regulatory requirements become more stringent so that there is strong incentive to encourage the use of less conservative parameter values.

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*In addition to the discussions of uncertainty included in this chapter, uncertainty is discussed in Appendices F and G. In Section F.5 of Appendix F, uncertainty analysis related to radiation dosimetry calculations is addressed. The uncertainties in risk estimates for radiogenic cancer are discussed in Section G.4 of Appendix G.

The consideration of uncertainty in individual parameter values used in environmental pathway models has been a subject of discussion in the technical community for more than a decade (Ba79a, Ba79b, Ho79, Mi79, Ru79, Sh79). However, the comprehensive consideration of overall uncertainty in environmental pathway, dosimetry and health impact analyses has begun to be addressed only recently (Ri83, Ru83).

When considering the uncertainty in the input parameters associated with environmental pathway calculations, the most common procedure has been to qualitatively consider the range of reported parameter values and to use judgement to select the "best" value to use for a particular application. More recently, there has been a tendency to statistically analyze the distribution of data for individual parameters and to choose a mean or median as the "best" value.

It appears that the most systematic mechanism for considering uncertainty in multiplicative chain models would be to include a probability distribution representing current uncertainty about parameter values in the input data and to run a sufficient number of cases (with parameter values for each case chosen by a suitable sampling procedure) such that the distribution of results can be evaluated. The results of this type of analysis could be considered in choosing an appropriate set of single-valued parameters to apply for regulatory calculations. Alternately, a decision might be made to perform the regulatory calculations probabilistically, then choose limits for a standard (or to perform calculations to see if a limit is met) at a specified confidence level. We believe the subject needs additional study to determine the most appropriate use of uncertainty analysis for standards setting applications; however, it is clear to us that a quantitative analysis of the uncertainties is most useful for focusing on important uncertainties for more intensive consideration.

EPA is presently involved in a re-evaluation of the methodology we apply in risk assessment calculations. In a speech at Princeton University on February 18, 1984, EPA Administrator William Ruckelshaus stated (Ru84), in proposing some principles for more reasonable discussions about risk, "First, we must insist on risk calculations being expressed as distributions of estimates and not as magic numbers that can be manipulated without regard to what they really mean. We must try to display more realistic estimates of risk to show a range of probabilities. To help do this we need new tools for quantifying and ordering sources of uncertainty and for putting them in perspective." Thus, at the highest level, EPA management is calling for an upgrading of our risk assessment methodology so that risk estimates can be more realistically expressed and the range of uncertainty in risk estimates more thoroughly quantified.

Most of the technical analyses discussed in this report were performed prior to the increase in emphasis on uncertainty in risk assessment calculations. The results presented in Chapter 6 were determined using point values for each parameter. In most cases, these point values were chosen after review of the range of values reported in the literature and were chosen to be near the mean or median value to avoid obtaining unrealistically conservative results. There are certain cases where conservative assumptions have been made, and these are discussed in the sections of the report describing the algorithms used in the computations (Chapter 3). Baes (Ba84) has published a very complete review and analysis of parameters used to predict the transport of radionuclides through agricultural pathways. For most radionuclides we considered, the river release mode was dominant and the agricultural pathways either dominated or were major pathways in determining the fatal cancers calculated for the river release mode. Default values from Baes report were used for many critical food pathway parameters and Baes states that these default values were chosen to be realistic rather than highly conservative. Since we were able to use default values based on Baes recent and extensive review of the literature, we believe that this significantly strengthens the analysis and causes it to be more realistic than would be the case if other sources of data were used.

The Envirosphere Company, a Division of Ebasco Services Incorporated, has recently completed a significant project (Ri83, ENV85) where they performed an uncertainty analysis for the input parameters of the river release mode algorithms. These are the algorithms used to derive the radionuclide release limits for Table 1 of 40CFR191 (EPA85c). Envirosphere reviewed the EPA river pathway models and identified the key uncertain input parameters for each river pathway. The key uncertain parameters were:

- soil-to-plant bioaccumulation factor (B_{iv1} and B_{iv2})[(a)]
- soil sink removal rate constant (λ_{Sn})
- fish reconcentration factor (CF_{np})
- resuspension factor (RF)
- sedimentation removal fraction*
- irrigation fraction (f_R)
- water-treatment removal fraction (f_{wt})*
- cow-to-milk transfer factor (F_{mn})[(a)]
- cow-to-beef transfer factor (F_{fn})[(a)]
- effective decay constant from vegetation (λ_{En})[(a)]
- dose conversion factors**
- risk conversion factors**

The uncertainties in these parameters were characterized by probability distributions which were propagated through the pathway models using a simulation technique which produced uncertainty distributions in the estimates of fatal cancers per unit radionuclide release to a river. The purpose of this work was to estimate the uncertainty associated with the EPA river release mode pathways to first-order and to identify the important uncertain parameters so that the values chosen for these parameters could be carefully re-evaluated. Preliminary Envirosphere analyses were presented to expert reviewers (Ri83) whose comments were used to improve some of the model input parameter uncertainty distributions for the final analysis, to better reflect the current state of knowledge about those factors.

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*These factors were set equal to 1.0 in the EPA calculations but were assigned a range of values for the Envirosphere calculations.

**These factors were combined in the EPA analysis into the fatal cancer risk factor (FCF_{np}).

[(a)] These factors are used in calculating the term RI_{np} in the EPA algorithms. They are defined and their application in the calculation of RI_{np} values is discussed in Section C.1 of Appendix C.

An example of the results of the Envirosphere uncertainty analysis is shown in Figure 7-1, which shows uncertainty distributions for the fatal cancers risk for Am-243 releases to a river*. Figure 7-1 shows a probability plot of the fatal cancers per 10,000 years per curie of Am-243 released to a river. The plots are generated by propagating the uncertainty distributions for the parameters listed above through the eight river pathway algorithms using stochastic simulation and a Latin Hypercube sampling technique. The details of the analysis are presented in the Envirosphere reports (Ri83, ENV85). Figure 7-1 indicates a 70 percent probability, considering parameter uncertainties, that the fatal cancers over 10,000 years per curie of Am-243 released to a river will be 5.72E-2, as calculated by EPA (Table 6-2), or less.

Similar calculations were performed for several other radionuclides listed in Table 1 of 40CFR191 and these calculations and the results are discussed in detail in the Envirosphere reports (Ri83, ENV85). The results

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*The 40CFR191 release limit for Am-243 is 100 Ci/10,000 yr/1000 MTHM.

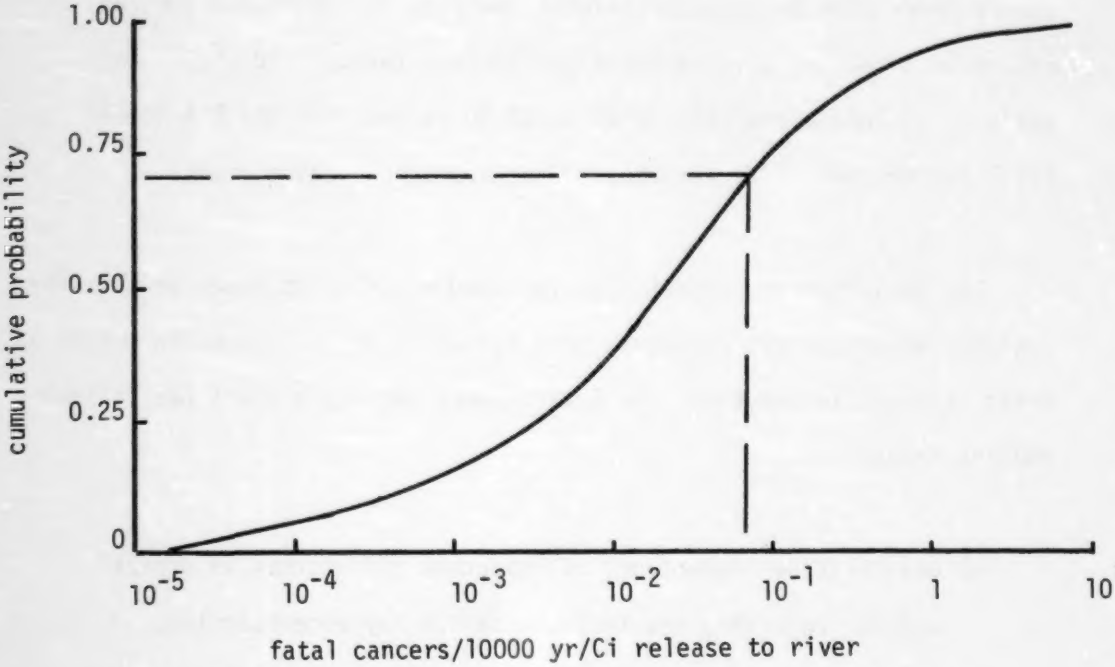

Figure 7-1. Cumulative probability distribution showing uncertainty about fatal cancer risk estimate for 1 curie of Am-243 released to a river (ENV85)

indicated, for all radionuclides analyzed except Np-237, a probability of 50 percent or more that the fatal cancers over 10,000 years per curie released to a river will be the number calculated by EPA or less. The exception was Np-237, which showed a 10 percent probability in the Envirosphere calculations. However, we believe there are equally supportable differences of expert opinion as to the appropriate shape of the parameter uncertainty distributions for the plant uptake factor (B_{iv}) and the fish concentration factor (CF_{np}). We requested that Envirosphere provide us an additional analysis for Np-237, using the alternate parameter uncertainty distributions for B_{iv} and CF_{np}. This analysis yielded a probability of about 50 percent for the EPA Np-237 fatal cancers per curie released to the accessible environment.

The Envirosphere project also included a number of model sensitivity analyses which are not discussed here but which are discussed in detail in their report. In addition, the Envirosphere reports discuss the following subject areas:

a. quantitative comparisons of important differences in expert opinion regarding how to characterize key uncertainties,
b. an examination of the sensitivity of dose and risk uncertainty results to the simulation technique used to propagate uncertainties, type of parameter uncertainty distributions used (e.g., uniform vs. log normal) and possible parameters correlation effects,
c. the relative importance of model parameter uncertainties with respect to the overall uncertainties in dose and risk estimates.

We used these results, along with suggestions by the EPA Science Advisory Board (SAB) Subcommittee (EPA84a), to identify those radionuclide release limits that warranted further consideration regarding the selection of appropriate values for important uncertain model parameters. Based upon this re-evaluation, some revisions were made to our input parameters in order to better reflect the current state of understanding regarding these parameters.

The Envirosphere report summarized a number of findings and conclusions which are quoted below. In some cases, we have added explanatory notes. We gave careful consideration to these findings and conclusions in the final analysis used as a basis for the 40CFR191 (Table 1) release limits.

Envirosphere Findings (Ri83)

1. "Uncertainty about the EPA model dose and risk outputs can span over nine orders of magnitude, depending on the radionuclide being analyzed."
2. "Most of the model's dose output uncertainty is attributable to uncertainties about the ingestion dose conversion factor, the soil-to-plant concentration factor (B_{iv1} and B_{iv2}), the soil root zone removal rate constant (λ_{Sn}) and the fraction of river flow used for irrigation. These pathway factors are all associated with the crop ingestion pathway, which is the largest dose contributing pathway of the eight included."

3. "Environmental pathway factor uncertainties are at least as significant as dose conversion factor uncertainty and risk coefficient uncertainty. In fact, many pathway factor uncertainties are larger contributors to effects uncertainty than are risk coefficient uncertainties in the EPA model, especially for high LET alpha emitters."

4. "Uncertainties about external dose conversion factors, transfer coefficients for forage to milk and forage to meat (F_{mn} and F_{fn}), the plant surface removal coefficient (λ_{En}) and the fraction of radionuclide removed by municipal water treatment are not important contributors to EPA model output uncertainty for this application."

5. "Soil resuspension factor uncertainty is only important for plutonium in this application of the EPA model, because plutonium is the only radionuclide specified in 40CFR191 which has a significant inhalation pathway dose contribution." (EPA Note: the air resuspension model has been modified, based on suggestions by the SAB subcommittee, and does not now contribute in a major way to the total dose and risk values for plutonium.)

6. "Several expert reviewers expressed dissatisfaction with the manner in which the EPA treated the removal of radioactivity from the soil root zone and resuspension of radioactive soil. In addition, removal of radionuclides by sedimentation processes in the river was considered to be important, especially for radionuclides with high binding coefficients, yet the EPA did not include sedimentation in their model. The treatment of these

processes in the EPA river pathways modeling warrants further consideration." (EPA Note: EPA is not aware of expert reviewer dissatisfaction with the model used to remove radioactivity from the soil root zone. Calculations were performed (see Appendix C, Section C.8) to show that recycling from the soil below the root zone into the river and back to the soil root zone was not significant for f_R values in the range applied by EPA ($f_R = 0.1$). Also, distribution coefficients (K_d's) have been revised to generally agree with those suggested by Baes (Ba84). As mentioned above, the resuspension model used by EPA has been revised in accordance with suggestions provided by the Science Advisory Board subcommittee. Several reviewers did suggest that EPA add sedimentation removal to our river model. There is no question that certain radionuclides deposit into sediments in slowly flowing rivers and lakes and, during the short-term, sedimentation lowers river water concentrations. However, in the long time period considered in these calculations, massive floods (100 and 1000 year floods, etc.), may occur so that much of the deposited activity may be resuspended from the sediment and distributed on farm land. For this reason and because of the difficulty involved in modelling sedimentation and subsequent resuspension during floods, we chose what we consider to be a conservation position and did not model radionuclide loss from river water due to sedimentation.)

7. "Possible correlations among model parameters can have a significant effect on uncertainty analysis results. Correlation

effects can increase or reduce model output uncertainties depending on the extent of correlation and the structure of the model. A lack of sufficient understanding about the processes which might result in parameters being correlated permits only a limited treatment of correlation effects."

8. "Identifying a specific repository site would considerably reduce model dose and effects output uncertainties since many key uncertainties are for site-related pathway factors; however, there would still be significant uncertainty contributions from dose conversion factors and the risk coefficient. Much of the overall uncertainty in dose estimation is contributed by site related pathway parameters, and, assuming time can be "frozen" in the assessment, once a specific site is identified measurements and data can be collected for these parameters. The time invarient assumption is necessary because to do otherwise eliminates the ability to make reasonably based quantitative judgments about model uncertainties. Also, the uncertainty characterization developed provides a structure for updating the analysis as the state of knowledge evolves and as changes occur."

Envirosphere Conclusions (Ri83)

Envirosphere stated the following conclusions concerning the use of probabilistic techniques for examining uncertainties in risk assessments:

1. "It is important to assure high quality in the input parameter uncertainty distributions used in an uncertainty analysis. The

validity of the results depends directly on the quality of the input uncertainty characterizations and the results are quite sensitive to the type of distribution assumed. Careful consideration must be given to the implications of using a particular probability distribution to represent a state of knowledge. Where valid differences of opinion exist, the sensitivity of model results to those alternative formulations should be tested."

2. "The type of analysis presented in this (Envirosphere) report focuses on important technical issues of the problem, provides a structure for updating the assessment as new information and understanding evolves and provides a basis for defensible and consistent standard-setting."

3. "The value of performing an analysis of uncertainties depends on the costs associated with being wrong about a risk management decision. These costs might include: (1) unexpected or unforeseen negative consequences; (2) misplaced or practically irreversible commitments of time, funds, manpower and capital; (3) adopting policies which are difficult to alter at a later date when new information becomes available, or (4) licensing, regulatory or legal complications and delays. For risk management applications an analysis of risk assessment uncertainties is, in effect, an analysis of investment uncertainties, and as such the uncertainty analysis should be coupled with a cost-benefit analysis. For the application examined in this study, the EPA should determine the relationship between cost of meeting various levels of release

> limits and the degree of certainty that those limits will achieve an acceptable level of population protection."

In summary, EPA is placing new emphasis on analyzing and discussing the uncertainty in our risk assessments. The basic calculations presented in this report were completed before the new emphasis was implemented. We have used point parameter values to calculate fatal cancers per unit radionuclide release and have generally used what we believe are realistic parameter values in the calculations, refined by the insights gained from the Envirosphere analysis and by suggestions by the SAB subcommittee. Taking into account the uncertainty analysis performed by Envirosphere helps to provide reasonable assurances that the EPA goal of 10 deaths in 10,000 years per 1000 MTHM is reflected by the release limits in Table 1 of 40CFR191.

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NOMENCLATURE

SUBSCRIPTS:

Unless specifically stated otherwise, these subscripts refer to the following:

a	aquifer
ar	aquifer-to-river
A	air
AL	air-over-land
AW	air-over-water
B	breathing
c, 14, and C-14	radionuclide C-14
D	radioactive decay
er	repository breaching event
f and FF	freshwater fish
g	air to ground
L	leaching (except refers to land surface pathway when used with variable t)
LL	directly to land
LS	land surface
n	nuclide
o	organ
p	pathway
ra	repository-to-aquifer
R	river (except refers to resuspension when used with variable λ)
s	soil
sw	surface water

SUBSCRIPTS (continued):

v	volcano/meteorite event
w	water or ocean
W	water
0	initial condition
1	upper ocean compartment
2	lower ocean compartment

SYMBOLS:

A	area of land irrigated (m^2)
A_L	land surface area for earth (m^2)
A_r	land area on which resuspended material is deposited (m^2)
A_w	ocean surface area for earth (m^2)
A_1	initial release rate of radionuclides into the ocean from the river (Ci/y)
CARCAN	risk factor to convert total body environmental dose commitment to environmental risk commitment for C-14 (fatal cancers/total body man rem)
CF_{np}	bioaccumulation factor for fish or shellfish for nuclide n and pathway p (Ci/kg per Ci/liter)
CP_p	the number of persons who can be fed from the quantity of crops (pathway p) raised annually on a unit area of land (man fed/m^2)
$D'(t)$	dose commitment rate as a function of time (rem/yr)
DTB_{14}	total body EDC as a function of integration time for C-14 releases (man rem/Ci released)
f_A	fraction of total release for the volcano/meteorite release mode which goes into air (dimensionless)

SYMBOLS (continued):

f_{AL}	fraction of total release which goes to air over land (dimensionless)
f_{AW}	fraction of total release which goes to air over water (dimensionless)
f_L	fraction of repository which is being leached (dimensionless)
f_{LL}	fraction of total release which goes directly onto land (dimensionless)
f_{LS}	fraction of the repository contents which is released to the land surface (dimensionless)
f_p	fraction of land used for various food crops for pathway p (dimensionless)
f_R	fraction of river flow used for irrigation (dimensionless)
f_{sw}	fraction of drinking water which is supplied from surface water sources (dimensionless)
f_{wt}	fraction of nuclide which passes through a water treatment plant and remains in potable water (dimensionless)
$F_n(t')$	quantity of radionuclide n deposited to ground per unit area integrated to time t' (Ci/m^2)
$F'_n(t)$	flux of nuclide n to ground as a function of time (Ci/m^2-yr)
FCF_{np}	fatal cancer risk factor for nuclide n and pathway p: for inhalation and ingestion (fatal cancers committed/Ci intake); for external air submersion (fatal cancers committed per Ci-y/m^3 integrated air concentration); for external ground contamination (fatal cancers committed per Ci-y/m^2 integrated ground concentration)
FHE_n	environmental risk commitment to the population for nuclide n, integrated to time t, for the release mode under consideration (fatal cancers committed)

SYMBOLS (continued):

FHE_{np}	environmental risk commitment to the population for nuclide n and pathway p, integrated to time t (fatal cancers committed)
FHE'_{np}	risk commitment rate to the population for nuclide n and pathway p (fatal cancers committed/yr)
GC_{np}	radionuclide specific factor used to correct ground surface fatal cancer risk factors to account for uniform distribution of radionuclides within the 15 cm soil root zone (dimensionless).
h_A	height of troposphere (m)
I_B	standard man breathing rate (m^3/yr)
I_f	freshwater fish annual consumption rate (kg/yr-person)
I_p	seafood annual consumption rate for pathway p (kg/yr)
I_W	annual individual water ingestion rate (liters/yr-person)
$N(D',t)$	size of the population exposed to dose commitment rate D'(t) at time t (persons)
P_{Ap}	population subjected to inhalation of radionuclides for pathway p (persons)
P_{FF}	population eating freshwater fish from the river (persons)
P_{Fp}	population eating food included in pathway p (persons)
P_p	population eating seafood for pathway p (persons)
P_R	population drinking water from river (persons)
PD_p	population density for pathway p (man/m^2)
$q_{1n}(t)$	quantity of radionuclide n in the upper compartment of the ocean for releases to ocean from rivers (Ci)
$q_{2n}(t)$	quantity of radionuclide n in the lower compartment of the ocean for releases to ocean from rivers (Ci)
$Q'(t')$	source term from ground to air at time t' (Ci/yr)

SYMBOLS (continued):

$Q_{An}(t')$	quantity of radionuclide n in the air-over-ocean compartment (Ci)
$Q'_{anp}(t)$	rate of entry of radionuclide n to an aquifer for pathway p (Ci/yr)
$Q'_D(t')$	source term from ground to air at time t' corrected for depletion (Ci/yr)
$Q_{Ln}(t')$	quantity of radionuclide n in the air-over-land compartment (Ci)
Q_{np}	total release of radionuclide n to the accessible environment into pathway p (Ci). For the land surface and air release mode, the release is instantaneous; for the river and ocean release mode, the release is integrated from time of placement in the repository out to time t.
Q_n	total release of radionuclide n to the accessible environment for the release mode under consideration (Ci) Note: $Q_n = Q_{np}$ since the releases considered for the different pathways within a release mode are all equal.
$Q'_{np}(t)$	rate of entry of radionuclide n into the river or ocean (Ci/y)
Q_{on}	initial inventory of radionuclide n in the repository (Ci)
$Q_{sn}(t')$	quantity of radionuclide n in the soil root zone (Ci)
$Q_{1n}(t')$	quantity of radionuclide n in the upper compartment of the ocean for releases to ocean from air (Ci)
$Q_{2n}(t')$	quantity of radionuclide n in the lower compartment of the ocean for releases to ocean from air (Ci)
r	radial distance from source of radionuclide to point of interest (m)
r_d	an empirical expression which is given in equation 3.3.2-4
R	river flow rate (liters/yr)
RF	resuspension factor (m^{-1})

SYMBOLS (continued):

RI_{np}	intake of nuclide n by an individual for the crop represented by pathway p and for a unit acute deposition to the surface (Ci intake per Ci/m^2 deposited on soil surface)
RIE_{np}	radionuclide intake or exposure for pathway p: for inhalation and ingestion (Ci intake); for external air submersion (Ci-y/m^3 exposure); for external ground contamination (Ci-y/m^2 exposure)
RIV'_{np}	annual risk commitment to an individual for nuclide n and pathway p (fatal cancers committed/yr)
SF_{1n}	sedimentation coefficient from upper layer of ocean for radionuclide n (yr^{-1})
SF_{2n}	sedimentation coefficient from lower layer of ocean for radionuclide n (yr^{-1})
SOF	shielding and occupancy factor (dimensionless)
t	time after placement in repository at which ERC is calculated (yr)
t'	time after release of radionuclides to the environment at which ERC is calculated (yr)
t"	dummy variable used for integration (yr)
t_{arn}	time for radionuclide n to travel from aquifer to river (yr)
t_{er}	time after placement in repository that radioactive material leaves the repository (yr)
t_L	time after placement in repository that the material comes to the surface for the land surface release mode (yr)
t_{ran}	time for radionuclide n to travel from repository to aquifer (yr)

SYMBOLS (continued):

t_{Rn}	time after placement in repository that radionuclide n enters the river or ocean (yr)
t_v	time after placement in the repository that the material enters into the land and air environment as the result of a volcano or meteorite (yr)
$T_{1/2}$	radiological half life (yr)
V_{AW}	volume of tropospheric air-over-water (m^3)
v_{gn}	deposition velocity from air to land surface (m/yr)
V_L	volume of tropospheric air over land (m^3)
v_{wn}	deposition velocity from air to ocean (m/yr)
V_1	volume of compartment 1 (upper compartment) of ocean (liters)
W	irrigation rate (liters/m^2-yr)
X_{1n}	concentration of radionuclide n in the upper compartment of the ocean for releases to the ocean from air (Ci/liter)
$X_{in}(r,t')$	air concentration at point r and time t' due to resuspension at initial source (Ci/m^3)
X_n	air concentration of radionuclide n due to resuspension from the ground surface at the location of interest and due to resuspension at the initial source and subsequent dispersion to the location of interest (Ci/m^3)
$(X/Q')_{r_n}$	atmospheric dispersion factor at the reference distance r_n (sec/m^3)
X_{Rn}	air concentration of radionuclide n at the center of a uniformly contaminated area having a surface concentration $\phi_n(t)$ due to resuspension of radionuclides from the ground surface (Ci/m^3)
z	"fitting" exponent in empirical equation for $X_{in}(r,t')$
γ_1	transfer rate coefficient from upper to lower ocean (yr^{-1})
γ_2	transfer rate coefficient from lower to upper ocean (yr^{-1})

SYMBOLS (continued):

λ_{Dn}	radioactive decay constant for nuclide n (yr^{-1})
λ_{gn}	rate constant for deposition from air to ground (yr^{-1})
λ_{Ln}	leaching rate constant from repository (yr^{-1})
λ_R	rate constant for resuspension of nuclides from soil to air (yr^{-1})
λ_{Sn}	rate constant for transfer of nuclides from available to unavailable soil for nuclide n (yr^{-1})
λ_{Tn}	$\lambda_R + \lambda_{Dn} + \lambda_{Sn}$
λ_{wn}	rate constant for deposition from air to ocean (yr^{-1})
ϕ'_{In}	rate of change with time of inventory of radionuclide n within the 15 cm root zone, expressed per unit surface area of soil (Ci/m^2-y).
ϕ_{In}	inventory of radionuclide n within the 15 cm root zone, expressed per unit surface area of soil (Ci/m^2)
ϕ'_n	rate of change with time of ground surface concentration (material within top 1 cm) for radionuclide n (Ci/m^2-y)
ϕ_n	ground surface concentration (material within top 1 cm) of radionuclide n as a function of time (Ci/m^2)

APPENDIX A: METHODS FOR CONSIDERATION OF DAUGHTER PRODUCT INGROWTH

For each parent radionuclide discussed in this report, the method for considering daughter product ingrowth is given in this Appendix (also see the discussion in section 4.2).

Radionuclide	Method for Consideration of Daughter Product Ingrowth
C-14 ($T_{1/2}$ = 5730 yr)	stable daughter - no action required
Ni-59 ($T_{1/2}$ = 8 E4 yr)	stable daughter - no action required
Sr-90 ($T_{1/2}$ = 28.1 yr)	assume Y-90 ($T_{1/2}$ = 64 hr) is in secular equilibrium and add Y-90 risk factors to those for Sr-90.
Zr-93 ($T_{1/2}$= 1.5 E6 yr)	assume Nb-93m ($T_{1/2}$ = 13.6 yr) is in secular equilibrium and add Nb-93m risk factors to those for Zr-93.
Tc-99 ($T_{1/2}$ = 2.1 E5 yr)	stable daughter - no action required.
Sn-126 ($T_{1/2}$ = 1 E5 yr)	use the decay scheme given by Kocher (Ko81a) for Sn-126. Assume Sb-126m and Sb-126 are in secular equilibrium with Sn-126. Then, using Kocher's decay scheme, add Sb-126m risk factors and 0.14·Sb-126 risk factors to those for Sn-126.
I-129 ($T_{1/2}$ = 1.7 E7 yr)	stable daughter - no action required.
Cs-135 ($T_{1/2}$ = 3 E6 yr)	stable daughter - no action required.

Radionuclide	Methods for Consideration of Daughter Product Ingrowth
Cs-137 ($T_{1/2}$ = 30 yr)	decays to Ba-137m 94.6 percent of the time. Assume that Ba-137m ($T_{1/2}$ = 2.55 min) is in secular equilibrium with Cs-137 and add 94.6 percent of dose factors for Ba-137m to the dose factors for Cs-137.
Sm-151 ($T_{1/2}$ = 87 yr)	stable daughter - no action required.
Pb-210 ($T_{1/2}$ = 22.3 yr)	assume Bi-210 ($T_{1/2}$ = 5.01 d) and Po-210 ($T_{1/2}$ = 138.4 d) are in secular equilibrium and add the risk factors for these two nuclides to those for Pb-210.
Ra-226 ($T_{1/2}$ = 1602 yr)	assume that all daughters of Ra-226 are in secular equilibrium and add their risk factors to the factors for Ra-226. The daughters assumed to be in secular equilibrium are Rn-222 ($T_{1/2}$ = 3.82 d), Po-218 ($T_{1/2}$ = 3.05 m), Pb-214 ($T_{1/2}$ = 26.8 m), Bi-214 ($T_{1/2}$ = 19.9 m), Po-214 ($T_{1/2}$ = 163.7 μs), Pb-210 ($T_{1/2}$ = 22.26 y), Bi-210 ($T_{1/2}$ = 5.01 d), and Po-210 ($T_{1/2}$ = 138.4 d).
Ra-228 ($T_{1/2}$ = 5.75 yr)	assume that all daughters of Ra-228 are in secular equilibrium and add their risk factors to the factors for Ra-228. The daughters assumed to be in secular equilibrium are Ac-228 ($T_{1/2}$ = 6.13 hr), Th-228 ($T_{1/2}$ = 1.91 yr), Ra-224 ($T_{1/2}$ = 3.62 d), Rn-220 ($T_{1/2}$ = 55.6 s), Po-216 ($T_{1/2}$ = 0.15 s), Pb-212 ($T_{1/2}$ = 10.64 hr), Bi-212 ($T_{1/2}$ = 60.6 m), 0.64 Po-212 ($T_{1/2}$ = 0.298 μs), 0.36 Tl-208 ($T_{1/2}$ = 3.05m).
Ac-227 ($T_{1/2}$ = 21.77 yr)	assume that all daughters of Ac-227 are in secular equilibrium and add their risk factors to the factors for Ac-227. The daughters assumed to be in secular equilibrium are Th-227 ($T_{1/2}$ = 18.7 d), Ra-223 ($T_{1/2}$ = 11.4 d), Rn-219 ($T_{1/2}$ = 3.96 s), Po-215 ($T_{1/2}$ = 1.78 ms), Pb-211 ($T_{1/2}$ = 36.1 m), Bi-211 ($T_{1/2}$ = 2.13 m), and Tl-207 ($T_{1/2}$ = 4.77 m).

Radionuclide	Methods for Consideration of Daughter Product Ingrowth
Th-229 ($T_{1/2}$ = 7.34E3 yr)	assume that all daughters of Th-229 are in secular equilibrium and add their risk factors to the factors for Th-229. The daughters assumed to be in secular equilibrium are Ra-225 ($T_{1/2}$ = 14.8 d), Ac-225 ($T_{1/2}$ = 10.0d), Fr-221 ($T_{1/2}$ = 4.8m), At-217 ($T_{1/2}$ = 32.3ms), Bi-213 ($T_{1/2}$ = 45.65m), Po-213 ($T_{1/2}$ = 4.2μs), and Pb-209 ($T_{1/2}$ = 3.25h).
Th-230 ($T_{1/2}$ = 7.7E4 yr)	the mean residence time of Th-230 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 185,200 yr. The first daughter is Ra-226 ($T_{1/2}$ = 1600 yr). We calculated that the Ra-226 concentration would peak at 0.92 of the initial Th-230 concentration so we assumed that all daughters of Th-230 are in secular equilibrium and add their risk factors to the factors for Th-230. The daughters assumed to be in secular equilibrium are Ra-226 ($T_{1/2}$ = 1600 yr), Rn-222 ($T_{1/2}$ = 3.82 d), Po-218 ($T_{1/2}$ = 3.05 m), Pb-214 ($T_{1/2}$ = 26.8 m), Bi-214 ($T_{1/2}$ = 19.9 m), Po-214 ($T_{1/2}$ = 163.7 μs), Pb-210 ($T_{1/2}$ = 22.26 yr), Bi-210 ($T_{1/2}$ = 5.01 d), and Po-210 ($T_{1/2}$ = 138.4 d).
Th-232 ($T_{1/2}$ = 1.405E10 yr)	assume that all daughters of Th-232 are in secular equilibrium and add their risk factors to the factors for Th-232. The daughters assumed to be in secular equilibrium are Ra-228 ($T_{1/2}$ = 5.75 yr), Ac-228 ($T_{1/2}$ = 6.13 h), Th-228 ($T_{1/2}$ = 1.91 yr), Ra-224 ($T_{1/2}$ = 3.62 d), Rn-220 ($T_{1/2}$ = 55.61 s), Po-216 ($T_{1/2}$ = 0.146 s), Pb-212 ($T_{1/2}$ = 10.64 hr), Bi-212 ($T_{1/2}$ = 60.55 m), 0.64 Po-212 ($T_{1/2}$ = 0.298 μs), and 0.36 Tl-208 ($T_{1/2}$ = 3.05 m).
Pa-231 ($T_{1/2}$ = 3.276E4 yr)	assume that all daughters of Pa-231 are in secular equilibrium and add their risk factors to the factors for Pa-231. The daughters assumed to be in secular equilibrium are Ac-227 ($T_{1/2}$ = 21.8 yr), Th-227 ($T_{1/2}$ = 18.7 d), Ra-223 ($T_{1/2}$ = 11.4 d), Rn-219 ($T_{1/2}$ = 3.96 s), Po-215 ($T_{1/2}$ = 1.78 ms), Pb-211 ($T_{1/2}$ = 36.1 m), Bi-211 ($T_{1/2}$ = 2.13 m), and Tl-207 ($T_{1/2}$ = 4.77 m).

Radionuclide	Methods for Consideration of Daughter Product Ingrowth
U-233 ($T_{1/2}$ = 1.592E5 yr)	the mean residence time of U-233 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 556 yr. The first daughter is Th-229 ($T_{1/2}$ = 7.34E3 yr). We calculated that the Th-229 concentration would peak at 0.05 of the initial U-233 concentration so we assume that all daughters of U-233 attain 5 percent of secular equilibrium and add 0.05 times their risk factors to the factors for U-233. The daughters assumed to reach 5 percent of secular equilibrium are Th-229 ($T_{1/2}$ = 7.34E3 yr), Ra-225 ($T_{1/2}$ = 14.8 d), Ac-225 ($T_{1/2}$ = 10.0 d), Fr-221 ($T_{1/2}$ = 4.8 m), At-217 ($T_{1/2}$ = 32.3 ms), Bi-213 ($T_{1/2}$ = 45.65 m), Po-213 ($T_{1/2}$ = 4.2 μs), and Pb-209 ($T_{1/2}$ = 3.25 h).
U-234 ($T_{1/2}$ = 2.5E5 yr)	the mean residence time of U-234 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 555 yr. The first daughter is Th-230 ($T_{1/2}$ = 8E 4 yr). We calculated that no significant levels of Th-230 would build up within the mean lifetime of U-234 in the soil root zone. Thus, there were no additions to the U-234 risk factors.
U-235 ($T_{1/2}$ = 7.038E8 yr)	assume Th-231 ($T_{1/2}$ = 25.52 hr) is in secular equilibrium with U-235. The mean residence time of U-235 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 556 yr. The second daughter is Pa-231 ($T_{1/2}$ = 3.276E4 yr). We calculated that the Pa-231 concentration would peak at 0.01 of the initial U-235 concentration so we assumed that Pa-231 and the remaining daughters attain 1 percent of secular equilibrium. Thus, we added 100 percent of the Th-231 risk factors and 1 percent of the remaining daughter risk factors to the factors for U-235. The daughters for which the additions were made are 1.0 Th-231 ($T_{1/2}$ = 25.5 h), 0.01 Pa-231 ($T_{1/2}$ = 3.276 E4 yr), 0.01 Ac-227 ($T_{1/2}$ = 21.77 yr), 0.01 Th-227 ($T_{1/2}$ = 18.7 d), 0.01 Ra-223 ($T_{1/2}$ = 11.4 d), 0.01 Rn-219 ($T_{1/2}$ = 3.96 s), 0.01 Po-215 ($T_{1/2}$ = 1.78 ms), 0.01 Pb-211 ($T_{1/2}$ = 36.1 m), 0.01 Bi-211 ($T_{1/2}$ = 2.13 m), and 0.01 Tl-207 (4.77 m).

Radionuclide	Methods for Consideration of Daughter Product Ingrowth
U-236 ($T_{1/2}$ = 2.3415E7 yr)	the mean residence time of U-236 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 556 yr. The first daughter is Th-232 ($T_{1/2}$ = 1.405E10 yr). We calculated that no significant levels of Th-232 would build up within the mean lifetime of U-236 in the soil root zone. There were no additions to the risk factors.
U-238 ($T_{1/2}$ = 4.468E9 yr)	the mean residence time of U-238 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 556 yr. The first and second daughters are Th-234 ($T_{1/2}$ = 24.1 d), and Pa-234m ($T_{1/2}$ = 1.17 m) which are assumed to be in secular equilibrium with U-238. The third daughter is U-234 ($T_{1/2}$ = 2.445E5 yr). We calculated that no significant levels of U-234 would build up within the mean lifetime of U-238 in the soil root zone. Thus, only the risk factors for Th-234 and Pa-234m were added to those for U-238.
Np-237 ($T_{1/2}$= 2.14 E6 yr)	assume Pa-233 ($T_{1/2}$ = 27d) is in secular equilibrium and add Pa-233 dose factors to those for Np-237. The mean residence time of Np-237 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 37 yr. The first long-lived daughter is U-233 ($T_{1/2}$ = 1.62E5 yr). We calculated that no significant levels of U-233 (and the daughters beyond) would build up within the mean lifetime of Np-237 in the soil root zone. Thus, there were no additions to Np-237 risk factors for U-233 and the daughters beyond U-233.
Pu-238 ($T_{1/2}$ = 86 y)	the mean residence time of Pu-238 in the soil root zone ($1/\lambda_s$), based on physical removal by leaching, is 5560 yr. The first daughter is U-234 ($T_{1/2}$ = 2.5E5 yr). We calculated that no significant levels of U-234 (and the daughters beyond) would build up in the soil root zone due to the decay of Pu-238. There were no additions to the Pu-238 risk factors.

Radionuclide	Methods for Consideration of Daughter Product Ingrowth
Pu-239 ($T_{1/2}$ = 2.4 E4 yr)	The mean residence time of Pu-239 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 5560 yr. The first daughter is U-235 ($T_{1/2}$ = 7.1E8 yr). We calculated that no significant levels of U-235 would build up within the mean lifetime of Pu-239 in the soil root zone. There were no additions to the Pu-239 risk factors.
Pu-240 ($T_{1/2}$ = 6580 yr)	The mean residence time of Pu-240 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 5560 yr. The first daughter is U-236 ($T_{1/2}$ = 2.4E7 yr). We calculated that no significant levels of U-236 would build up within the mean lifetime of Pu-240 in the soil root zone. There were no additions to the Pu-240 risk factors.
Pu-241 ($T_{1/2}$ = 14.4 yr)	The mean residence time of Pu in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 5556 yr. This is far longer than it would take the Pu-241 to disappear due to radioactive decay. The first daughter is Am-241 ($T_{1/2}$ = 432.2 yr). We calculated that the Am-241 concentration would peak at 0.03 of the initial Pu-241 concentration. Np-237 ($T_{1/2}$ = 2.14E6 yr) is the second daughter and there would be no significant buildup in the soil root zone. The Pu-241 risk factors were augmented by the addition of 0.03 times the risk factors for Am-241.
Pu-242 ($T_{1/2}$ = 3.8 E5 yr)	The mean residence time of Pu-242 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 5560 yr. The first daughter is U-238 ($T_{1/2}$ = 4.5E9 yr). We calculated that no significant levels of U-238 would build up within the mean lifetime of Pu-242 in the soil root zone. There were no additions to the Pu-242 risk factors.
Am-241 ($T_{1/2}$ = 458 yr)	The mean residence time of Am-241 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 862 yr. The first daughter is Np-237 ($T_{1/2}$ = 2.1E6 yr). We calculated that no significant levels of Np-237 would build up within the mean lifetime of Am-241 in the soil root zone. There were no additions to the Am-241 risk factors.

Radionuclide	Methods for Consideration of Daughter Product Ingrowth
Am-243 ($T_{1/2}$ = 7950 yr)	Assume Np-239 ($T_{1/2}$ = 2.3 d) is in secular equilibrium and add Np-239 risk factors to those for Am-243. The mean residence time of Am-243 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 862 yr. The first long-lived daughter is Pu-239 ($T_{1/2}$ = 2.4E4 yr). We calculated that no significant levels of Pu-239 would build up within the mean lifetime of Am-243 in the soil root zone. There were no additions to the Am-243 risk factors for Pu-239 and the daughters beyond Pu-239.
Cm-245 ($T_{1/2}$ = 8.5E3 yr)	The mean residence time of Cm-245 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 2470 yr. The first and second daughters are Pu-241 ($T_{1/2}$ = 14.4 yr) and Am-241 ($T_{1/2}$ = 432.2 yr) which are assumed to be in secular equilibrium with Cm-245. The third daughter is Np-237 ($T_{1/2}$ = 2.14E6 yr). We calculated that no significant levels of Np-237 would build up within the mean lifetime of Cm-245 in the soil root zone. Thus, only the risk factors for Pu-241 and Am-241 were added to those for Cm-245.
Cm-246 ($T_{1/2}$ = 4.75E3 yr)	The mean residence time of Cm-246 in the soil root zone ($1/\lambda_s$), based on removal by leaching, is 2470 yr. The first daughter is Pu-242 ($T_{1/2}$ = 3.758E5 yr). We calculated that no significant levels of Pu-242 would build up within the mean lifetime of Cm-246 in the soil root zone. There were no additions to the Cm-246 risk factors.

APPENDIX B: FATAL CANCER RISK FACTORS

The fatal cancer risk factors which were applied in these analyses are listed in Table B-1. Table B-1 lists the risk factors for the parent radionuclide and for daughter radionuclides which can grow in (and be inhaled, ingested, or cause external exposure in addition to the parent) during the mean lifetime of the parent in the soil root zone. For inhalation and ingestion, all risk factors listed in Table B-1 incorporate the ingrowth and dynamics of daughters in the body after intake of a radionuclide.

The radionuclides shown in Table B-1 have risk factors whose magnitude exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which could grow in during the mean lifetime of the parent in the soil root zone. Included in Table B-1 is the clearance class and value for the gut-to-blood transfer fraction, f_1, as applicable. The source of these risk factors is a run of the EPA RADRISK computer code using the V8401BD version of the input data file (Ne84).

An explanation of the methodology used to derive these risk factors is given in Chapter 4*. For the inhalation and ingestion pathways two categories of fatal cancer risk factors were calculated which were designated Inhalation 1, Inhalation 2, Ingestion 1, and Ingestion 2. Class Y risk factors were used for the Inhalation 1 category for all nuclides where they were available and Class W risk factors were used for the Inhalation 2 category where they were available. However, for some radionuclides, only Class D or Class D and Class W inhalation risk factors were available. In these cases, the factors for the least soluble clearance class available were applied. For example, if inhalation risk factors were available for Class D and Class W but not for Class Y, the Class W factors were listed in both the Inhalation 1 and Inhalation 2 categories. For the Ingestion 1 and Ingestion 2 categories, risk factors were used which were derived using the same absorption fraction from gut-to-blood (f_1) as was used for the Inhalation 1 and Inhalation 2 categories, respectively.

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*The technical basis for the EPA RADRISK computer code and data base in discussed in detail in Appendices F, G and H.

TABLE B-1
Inhalation 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

	********************************* Daughters ********************************	
Parent	Nuclide, Clearance Class, f_1** Risk Factor	Parent + Daughters
C-14, -, -, 3.053E-3		3.05E-3
Ni-59, W, 5E-2 4.761E-1		4.76E-1
Sr-90, Y, 1E-2 4.479E2		4.52E2
Zr-93, Y, 2E-3 1.269E1	Nb-93m, Y, 1E-2 1.449E1	2.72E1
Tc-99, W, 8E-1 6.120E0		6.12E0
Sn-126, W, 2E-2 5.637E1	(.14)Sb-126, W, 1E-2 8.310E-1	5.72E1
I-129, D, 9.5E-1 1.605E+1		1.61E1
Cs-135, D, 9.5E-1 1.266E0		1.27E0

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

** For each nuclide listed for inhalation pathways, the first line includes the nuclide symbol and the clearance class and value for f_1 used to derive the risk factor. The second line lists the risk factor.

TABLE B-1 (Continued)
Inhalation 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	****************************** Daughters ******************************			Parent + Daughters
	Nuclide, Clearance Class, f_1 Risk Factor			
Cs-137, D, 9.5E-1 8.486E0				8.49E0
Sm-151, W, 3E-4 5.271E0				5.27E0
Pb-210, Y, 2E-1 1.715E4	Bi-210, Y, 5E-2 1.910E2	Po-210, Y, 1E-1 5.311E3		2.27E4
Ra-226, Y, 2E-1 2.116E4	Pb-210, Y, 2E-1 1.715E4	Po-210, Y, 1E-1 5.311E3		4.38E4
Ra-228, Y, 2E-1 1.841E4	Th-228, Y, 2E-4 6.030E4	Ra-224, Y, 2E-1 9.901E2		7.98E4
Ac-227, Y, 1E-3 6.339E4	Th-227, Y, 2E-4 3.826E3	Ra-223, W, 2E-1 2.443E3		6.97E4
Th-229, Y, 2E-4 6.138E4	Ra-225, W, 2E-1 1.211E3	Ac-225, Y, 1E-3 1.883E3		6.45E4
Th-230, Y, 2E-4 2.510E4	Ra-226, Y, 2E-1 2.116E4	Pb-210, Y, 2E-1 1.715E4	Po-210, Y, 1E-1 5.311E3	6.89E4

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Inhalation 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	********************************* Daughters *********************************			Parent + Daughters
	Nuclide, Clearance Class, f_1 Risk Factor			
Th-232, Y, 2E-4 2.475E4	Ra-228, Y, 2E-1 1.841E4	Th-228, Y, 2E-4 6.030E4	Ra-224, Y, 2E-1 9.901E2	1.05E5
Pa-231, Y, 1E-3 3.287E4	Ac-227, Y, 1E-3 6.399E4	Th-227, Y, 2E-4 3.826E3	Ra-223, W, 2E-1 2.443E3	1.03E5
U-233, Y, 2E-3 2.095E4	(.05)Th-229, Y, 2E-4 3.069E3			2.42E4
U-234, Y, 2E-3 2.070E4				2.07E4
U-235, Y, 2E-3 1.922E4	(.01)Pa-231, Y, 1E-3 3.287E2	(.01)Ac-227, Y, 1E-3 6.339E2		2.03E4
U-236, Y, 2E-3 1.959E4				1.96E4
U-238, Y, 2E-3 1.854E4				1.85E4
Np-237, Y, 1E-3 2.888E4				2.89E4

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Inhalation 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	********************************** Daughters ********************************** Nuclide, Clearance Class, f_1 Risk Factor	Parent + Daughters
Pu-238, Y, 1E-3 3.134E4		3.13E4
Pu-239, Y, 1E-4 3.093E4		3.09E4
Pu-240, Y, 1E-4 3.092E4		3.09E4
Pu-241, Y, 1E-3 2.073E2	(.03)Am-241, Y, 1E-3 9.798E2	1.19E3
Pu-242, Y, 1E-4 2.936E4		2.94E4
Am-241, Y, 1E-3 3.266E4		3.27E4
Am-243, Y, 1E-3 3.193E4		3.19E4
Cm-245, Y, 1E-3 3.249E4	Am-241, Y, 1E-3 3.266E4	6.54E4
Cm-246, Y, 1E-3 3.263E4		3.26E4

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Inhalation 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	******************************** Daughters ******************************** Nuclide, Clearance Class, f_1 Risk Factor	Parent + Daughters
C-14, -, - 3.053E-3		3.05E-3
Ni-59, W, 5E-2 4.761E-1		4.76E-1
Sr-90, D, 3E-1 4.833E1	Y-90, W, 1E-4 3.556E0	5.19E1
Zr-93, W, 2E-3 4.819E0	Nb-93m, W, 1E-2 1.776E0	6.60E0
Tc-99, W, 8E-1 6.120E0		6.12E0
Sn-126, W, 2E-2 5.637E1	(0.14)Sb-126, W, 1E-2 8.310E-1	5.72E1
I-129, D, 9.5E-1 1.605E1		1.61E1
Cs-135, D, 9.5E-1 1.266E0		1.27E0

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Inhalation 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	********************************* Daughters *******************************			Parent + Daughters
	Nuclide, Clearance Class, f_1 Risk Factor			
Cs-137, D, 9.5E-1 8.486E0				8.49E0
Sm-151, W, 3E-4 5.271E0				5.27E0
Pb-210, W, 2E-1 9.961E2	Bi-210, W, 5E-2 6.182E1	Po-210, W, 1E-1 1.930E3		2.99E3
Ra-226, W, 2E-1 2.331E3	Pb-210, W, 2E-1 9.961E2	Bi-210, W, 5E-2 6.182E1	Po-210, W, 1E-1 1.930E3	5.33E3
Ra-228, W, 2E-1 4.754E2	Th-228, W, 2E-4 1.434E4	Ra-224, W, 2E-1 9.443E2		1.58E4
Ac-227, W, 1E-3 3.232E4	Th-227, W, 2E-4 2.667E3	Ra-223, W, 2E-1 2.443E3		3.74E4
Th-229, W, 2E-4 2.528E4	Ra-225, W, 2E-1 1.211E3	Ac-225, W, 1E-3 1.715E3		2.82E4
Th-230, W, 2E-4 1.514E4	Ra-226, W, 2E-1 2.331E3	Pb-210, W, 2E-1 9.961E2	Po-210, W, 1E-1 1.930E3	2.05E4

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Inhalation 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	Daughters				Parent + Daughters
	Nuclide, Clearance Class, f_1 Risk Factor				
Th-232, W, 2E-4 1.358E4	Ra-228, W, 2E-1 4.754E2	Th-228, W, 2E-4 1.434E4	Ra-224, W, 2E-1 9.443E2		2.94E4
Pa-231, W, 1E-3 2.442E4	Ac-227, W, 1E-3 3.232E4	Th-227, W, 2E-4 2.667E3	Ra-223, W, 2E-1 2.443E3		6.19E4
U-233, W, 2E-1 2.291E3	(.05)Th-229, W, 2E-4 1.264E3	(.05)Ra-225, W, 2E-1 6.055E1	(.05)Ac-225, W, 1E-3 8.575E1		3.70E3
U-234, W, 2E-1 2.263E3					2.26E3
U-235, W, 2E-1 2.104E3	(.01)Pa-231, W, 1E-3 2.442E2	(.01)Ac-227, W, 1E-3 3.232E2	(.01)Th-227, W, 2E-4 2.667E1	(.01)Ra-223, W, 2E-1 2.443E1	2.72E3
U-236, W, 2E-1 2.141E3					2.14E3
U-238, W, 2E-1 2.026E3					2.04E3
Np-237, W, 1E-3 2.464E4					2.46E4

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Inhalation 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

	********************************** Daughters *********************************		
Parent	Nuclide, Clearance Class, f_1 Risk Factor		Parent + Daughters
Pu-238, W, 1E-3 2.491E4			2.49E4
Pu-239, W, 1E-3 2.648E4			2.65E4
Pu-240, W, 1E-3 2.645E4			2.65E4
Pu-241, W, 1E-3 4.071E2	(.03)Am-241, W, 1E-3 8.247E2		1.23E3
Pu-242, W, 1E-3 2.515E4			2.52E4
Am-241, W, 1E-3 2.749E4			2.75E4
Am-243, W, 1E-3 2.727E4			2.73E4
Cm-245, W, 1E-3 2.794E4	Pu-241, W, 1E-3 4.071E2	Am-241, W, 1E-3 2.749E4	5.58E4
Cm-246, W, 1E-3 2.782E4			2.78E4

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	******************************** Daughters ********************************			Parent + Daughters
	Nuclide, f_1** Risk Factor			
C-14, - 4.321E-1				4.32E-1
Ni-59, 5E-2 3.761E-2				3.76E-2
Sr-90, 1E-2 1.452E0	Y-90, 1E-4 8.391E-1			2.29E0
Zr-93, 2E-3 8.459E-2	Nb-93m, 1E-2 4.213E-2			1.27E-1
Tc-99, 8E-1 5.374E-1				5.37E-1
Sn-126, 2E-2 1.895E0	Sb-126m, 1E-2 2.859E-2	(.14)Sb-126, 1E-2 1.182E-1		2.04E0
I-129, 9.5E-1 2.407E1				2.41E1
Cs-135, 9.5E-1 1.819E0				1.82E0

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

** For each nuclide listed for ingestion pathways, the first line includes the nuclide symbol and the value for f_1 used to derive the risk factor. The second line lists the risk factor.

TABLE B-1 (Continued)
Ingestion 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	******************************** Daughters ********************************			Parent + Daughters
	Nuclide, f_1 Risk Factor			
Cs-137, 9.5E-1 1.241E+1				1.24E1
Sm-151, 3E-4 3.461E-2				3.46E-2
Pb-210, 2E-1 3.409E2	Po-210, 1E-1 7.181E1			4.13E2
Ra-226, 2E-1 7.783E1	Pb-210, 2E-1 3.409E2	Po-210, 1E-1 7.181E1		4.91E2
Ra-228, 2E-1 5.474E1	Th-228, 2E-4 1.053E1	Ra-224, 2E-1 2.828E1	Pb-212, 2E-1 3.265E0	9.71E1
Ac-227, 1E-3 2.335E2	Ra-223, 2E-1 4.938E1			2.85E2
Th-229, 2E-4 3.526E1	Ra-225, 2E-1 4.419E1	Ac-225, 1E-3 5.883E0		8.55E1
Th-230, 2E-4 2.198E1	Ra-226, 2E-1 7.783E1	Pb-210, 2E-1 3.409E2	Po-210, 1E-1 7.181E1	5.13E2

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	********************************** Daughters ******************************** Nuclide, f_1 Risk Factor				Parent + Daughters
Th-232, 2E-4 1.985E1	Ra-228, 2E-1 5.474E1	Th-228, 2E-4 1.053E1	Ra-224, 2E-1 2.828E1	Pb-212, 2E-1 3.265E0	1.17E2
Pa-231, 1E-3 1.827E2	Ac-227, 1E-3 2.335E2	Ra-223, 2E-1 4.938E1			4.67E2
U-233, 2E-3 9.387E-1	(.05)Th-229, 2E-4 1.763E0	(.05)Ra-225, 2E-1 2.210E0	(.05)Ac-225,1E-3 2.942E-1		5.21E0
U-234, 2E-3 9.382E-1					9.38E-1
U-235, 2E-3 1.081E0	Th-231, 2E-4 1.083E-1	(.01)Pa-231, 1E-3 1.827E0	(.01)Ac-227, 1E-3 2.335E0	(0.01)Ra-223, 2E-1 4.938E-1	5.86E0
U-236, 2E-3 8.864E-1					8.86E-1
U-238, 2E-3 9.361E-1	Th-234, 2E-4 1.049E0				1.99E0
Np-237, 1E-3 1.860E2					1.86E2

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 1: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	********************************** Daughters *********************************		Parent + Daughters
	Nuclide, f_1 Risk Factor		
Pu-238, 1E-3 1.856E2			1.86E2
Pu-239, 1E-4 2.043E1			2.04E1
Pu-240, 1E-4 2.041E1			2.04E1
Pu-241, 1E-3 3.361E0	(.03)Am-241, 1E-3 6.207E0		9.57E0
Pu-242, 1E-4 1.941E1			1.94E1
Am-241, 1E-3 2.069E2			2.07E2
Am-243, 1E-3 2.059E2			2.06E2
Cm-245, 1E-3 2.111E2	Pu-241, 1E-3 3.361E0	Am-241, 1E-3 2.069E2	4.21E2
Cm-246, 1E-3 2.098E2			2.10E2

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

	********************************** Daughters ********************************		
Parent	Nuclide, f_1 Risk Factor		Parent + Daughters
C-14, - 4.321E-1			4.32E-1
Ni-59, 5E-2 3.761E-2			3.76E-2
Sr-90, 3E-1 2.762E1	Y-90, 1E-4 8.391E-1		2.85E1
Zr-93, 2E-3 8.459E-2	Nb-93m, 1E-2 4.213E-2		1.27E-1
Tc-99, 8E-1 5.374E-1			5.37E-1
Sn-126, 2E-2 1.895E0	Sb-126m, 1E-2 2.859E-2	(.14)Sb-126, 1E-2 1.182E-1	2.04E0
I-129, 9.5E-1 2.407E+1			2.41E+1
Cs-135, 9.5E-1 1.819E0			1.82E0

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	******************************** Daughters ******************************** Nuclide, f_1 Risk Factor			Parent + Daughters
Cs-137, 9.5E-1 1.241E1				1.24E1
Sm-151, 3E-4 3.461E-2				3.46E-2
Pb-210, 2E-1 3.409E2	Po-210, 1E-1 7.181E1			4.13E2
Ra-226, 2E-1 7.783E1	Pb-210, 2E-1 3.409E2	Po-210, 1E-1 7.181E1		4.91E2
Ra-228, 2E-1 5.474E1	Th-228, 2E-4 1.053E1	Ra-224, 2E-1 2.828E1	Pb-212, 2E-1 3.265E0	9.71E1
Ac-227, 1E-3 2.335E2	Ra-223, 2E-1 4.938E1			2.85E2
Th-229, 2E-4 3.526E1	Ra-225, 2E-1 4.419E1	Ac-225, 1E-3 5.883E0		8.55E1
Th-230, 2E-4 2.198E1	Ra-226, 2E-1 7.783E1	Pb-210, 2E-1 3.409E2	Po-210, 1E-1 7.181E1	5.13E2

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

	********************************* Daughters *********************************				
Parent	Nuclide, f_1 Risk Factor				Parent + Daughters
Th-232, 2E-4 1.985E1	Ra-228, 2E-1 5.474E1	Th-228, 2E-4 1.053E1	Ra-224, 2E-1 2.828E1	Pb-212, 2E-1 3.265E0	1.17E2
Pa-231, 1E-3 1.827E2	Ac-227, 1E-3 2.335E2	Ra-223, 2E-1 4.938E1			4.67E2
U-233, 2E-1 4.646E1	(.05)Th-229, 2E-4 1.763E0	(.05)Ra-225, 2E-1 2.210E0			5.07E1
U-234, 2E-1 4.605E1					4.61E1
U-235, 2E-1 4.546E1	(.01)Pa-231, 1E-3 1.827E0	(.01)Ac-227, 1E-3 2.335E0	(.01)Ra-223, 2E-1 4.938E-1		5.02E1
U-236, 2E-1 4.353E1					4.35E1
U-238, 2E-1 4.735E1	Th-234, 2E-4 1.049E0				4.84E1
Np-237, 1E-3 1.860E2					1.86E2

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ingestion 2: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci intake)

Parent	********************************** Daughters ******************************** Nuclide, f_1 Risk Factor		Parent + Daughters
Pu-238, 1E-3 1.856E2			1.86E2
Pu-239, 1E-3 1.997E2			2.00E2
Pu-240, 1E-3 1.994E2			1.99E2
Pu-241, 1E-3 3.361E0	(.03)Am-241, 1E-3 6.207E0		9.57E0
Pu-242, 1E-3 1.897E2			1.90E2
Am-241, 1E-3 2.069E2			2.07E2
Am-243, 1E-3 2.059E2			2.06E2
Cm-245, 1E-3 2.111E2	Pu-241, 1E-3 3.361E0	Am-241, 1E-3 2.069E2	4.21E2
Cm-246, 1E-3 2.098E2			2.10E2

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Air Submersion: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^3 exposure)

Parent	******************************** Daughters ********************************** Nuclide** Risk Factor		Parent + Daughters
C-14 0			0
Ni-59 4.100E-2			4.10E-2
Sr-90 0			0
Zr-93 0	Nb-93m 1.232E-1		1.23E-1
Tc-99 5.974E-4			5.97E-4
Sn-126 5.395E1	Sb-126m 1.993E3	(.14)Sb-126 4.913E2	2.54E3
1-129 7.572E0			7.57E0
Cs-135 0			0

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

** For each nuclide listed for air submersion and for ground deposition, the first line includes the nuclide symbol and the second line lists the risk factor.

TABLE B-1 (Continued)
Air Submersion: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^3 exposure)

Parent	******************************** Daughters **********************************					Parent + Daughters
	Nuclide Risk Factor					
Cs-137 0	(.946)Ba-137m 7.179E2					7.18E2
Sm-151 8.148E-4						8.15E-4
Pb-210 1.332E0						1.33E0
Ra-226 8.183E0	Pb-214 3.033E2	Bi-214 2.032E3				2.35E3
Ra-228 6.814E-9	Ac-228 1.202E3	Pb-212 1.763E2	Bi-212 2.392E2	(.36)Tl-208 1.793E3		3.43E3
Ac-227 1.445E-1	Th-227 1.258E2	Ra-223 1.589E2	Rn-219 7.019E1	Pb-211 6.464E1	Bi-211 5.786E1	4.81E2
Th-229 9.923E1	Ra-225 6.457E0	Ac-225 1.574E1	Fr-221 3.748E1	Bi-213 1.711E2		3.30E2
Th-230 4.350E-1	Pb-214 3.033E2	Bi-214 2.032E3				2.35E3

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Air Submersion: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^3 exposure)

Parent	****************************** Daughters *********************************					Parent + Daughters
			Nuclide Risk Factor			
Th-232 2.019E-1	Ac-228 1.202E3	Pb-212 1.763E2	Bi-212 2.392E2	(.36)Tl-208 1.793E3		3.43E3
Pa-231 3.629E1	Th-227 1.258E2	Ra-223 1.589E2	Rn-219 7.019E1	Pb-211 6.464E1	Bi-211 5.786E1	5.17E2
U-233 2.734E-1	(.05)Th-229 4.962E0	(.05)Ra-225 3.229E-1	(.05)Ac-225 7.870E-1	(.05)Fr-221 1.874E0	(.05)Bi-213 8.555E0	1.68E1
U-234 1.631E-1						1.63E-1
U-235 1.833E2	Th-231 1.285E1					2.01E2
U-236 1.269E-1						1.27E-1
U-238 1.085E-1	Th-234 8.635E0	Pa-234m 1.485E1				2.36E1
Np-237 2.581E1	Pa-233 2.575E2					2.83E2

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Air Submersion: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^3 exposure)

	******************************** Daughters **********************************	
Parent	Nuclide Risk Factor	Parent + Daughters
Pu-238 8.802E-2		8.80E-2
Pu-239 9.058E-2		9.06E-2
Pu-240 8.634E-2		8.63E-2
Pu-241 0	(.03)Am-241 5.982E-1	5.98E-1
Pu-242 7.362E-2		7.36E-2
Am-241 1.994E1		1.99E1
Am-243 5.592E1	Np-239 1.986E2	2.55E2
Cm-245 8.242E1	Am-241 1.994E1	1.02E2
Cm-246 6.805E-2		6.81E-2

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ground Deposition: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^2 exposure)

Parent	************ Daughters ************ Nuclide Risk Factor		Parent + Daughters
C-14 0			0
Ni-59 8.871E-3			8.87E-3
Sr-90 0			0
Zr-93 0	Nb-93m 1.885E-2		1.89E-2
Tc-99 1.409E-5			1.41E-5
Sn-126 1.354E0	Sb-126m 4.001E1	(.14)Sb-126 9.769E0	5.11E1
I-129 3.977E-1			3.98E-1
Cs-135 0			0

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ground Deposition: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^2 exposure)

Parent	*********************************	********** Daughters	**********	*********************	**********	Parent + Daughters
		Nuclide Risk Factor				
Cs-137 0	(.946)Ba-137m 1.432E1					1.43E1
Sm-151 9.425E-5						9.43E-5
Pb-210 6.067E-2						6.09E-2
Ra-226 1.811E-1	Pb-214 6.528E0	Bi-214 3.521E1				4.20E1
Ra-228 1.427E-8	Ac-228 2.264E1	Ra-224 2.670E-1	Pb-212 3.927E0	Bi-212 4.436E0	(.36)Tl-208 2.745E1	5.88E1
Ac-227 4.661E-3	Th-227 2.825E0	Ra-223 3.583E0	Rn-219 1.509E0	Pb-211 1.293E0	Bi-211 1.242E0	1.05E1
Th-229 2.390E0	Ra-225 2.873E-1	Ac-225 3.806E-1	Fr-221 8.267E-1	Bi-213 3.577E0		7.46E0
Th-230 1.928E-2	Pb-214 6.528E0	Bi-214 3.521E1				4.20E1

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ground Deposition: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^2 exposure)

	******************************** Daughters ********************************					
Parent			Nuclide Risk Factor			Parent + Daughters
Th-232 1.383E-2	Ac-228 2.264E1	Ra-224 2.670E-1	Pb-212 3.927E0	Bi-212 4.436E0	(.36)T1-208 2.745E1	5.88E1
Pa-231 8.488E-1	Th-227 2.825E0	Ra-223 3.583E0	Rn-219 1.509E0	Pb-211 1.293E0	Bi-211 1.242E0	1.14E1
U-233 1.076E-2	(.05)Th-229 1.195E-1	(.05)Ra-225 1.437E-2	(.05)Ac-225 1.903E-2	(.05)Fr-221 4.134E-2	(.05)Bi-213 1.789E-1	3.84E-1
U-234 1.634E-2						1.63E-2
U-235 4.080E0	Th-231 4.089E-1	(.01)Ra-223 3.583E-2				4.60E0
U-236 1.476E-2						1.48E-2
U-238 1.300E-2	Th-234 2.225E-1	Pa-234m 2.815E-1				5.17E-1
Np-237 7.105E-1	Pa-233 5.681E0					6.39E0

* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

TABLE B-1 (Continued)
Ground Deposition: Fatal cancer risk conversion factors for parent and significant* daughters
(fatal cancers committed per Ci-y/m^2 exposure)

Parent	****************************** Daughters ****************************** Nuclide Risk Factor	Parent + Daughters
Pu-238 1.681E-2		1.68E-2
Pu-239 7.642E-3		7.64E-3
Pu-240 1.610E-2		1.61E-2
Pu-241 0	(.03)Am-241 1.871E-2	1.87E-2
Pu-242 1.337E-2		1.34E-2
Am-241 6.236E-1		6.24E-1
Am-243 1.437E0	Np-239 4.511E0	5.95E0
Cm-245 1.959E0	Am-241 6.236E-1	2.58E0
Cm-246 1.411E-2		1.41E-2

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* Daughters for which the risk factor exceeds 1 percent of the maximum risk factor for any nuclide in the decay chain which can grow in while the parent exists in the accessible environment.

APPENDIX C: COMPUTATIONAL DETAILS

In this appendix, we will provide the details of the derivation of the values used for several parameters which were discussed in Chapter 5. A subsection of this appendix has been set aside for each parameter to be discussed.

C.1 Curie Intake per Unit Area Deposition (RI_{np})

The parameter, RI_{np}, expresses the individual intake of a radionuclide (n) per unit acute deposition to the soil surface for a food crop (p). The associated units attached to this parameter are Ci intake per Ci/m^2 deposited to the soil. The algorithms used to compute values for RI_{np} were similar to those given in the AIRDOS-EPA computer code manual (Mo79) and those applied by NRC in Regulatory Guide 1.109(NRC77). The computational techniques can be traced back to the HERMES computer code (F171) which was prepared by the Pacific Northwest Laboratories in the early 1970's.

For food crops consumed directly by humans, we can express RI_{np} using the rate of consumption of vegetation by a human (U^p and U^L), the concentration of radionuclides in the vegetation (C_n^p and C_n^L), and the deposition rate of radionuclides to the ground (d_n) as

$$RI_{np} = \frac{U^p\ C_n^p}{d_n} + \frac{U^L\ C_n^L}{d_n} \qquad \text{(C-1)}$$

where

U^p and U^L = quantity of vegetation consumed by a human receptor per unit time for produce (kg wet/yr) and leafy vegetables (kg dry/yr), respectively,

C_n^P and C_n^L = concentration of nuclide n in and on vegetation for produce (Ci/kg wet) and leafy vegetables (Ci/kg dry), respectively,

d_n = the deposition rate of nuclide n onto the ground (Ci/m^2-yr).

Note that the dimensions for RI_{np} from equation C-1 are Ci intake/yr per Ci deposited/m^2-yr whereas the dimensions we desire are Ci intake over all time per Ci deposited (total)/m^2. It can be shown that the ratio of the equilibrium intake rate to a continuous deposition rate (Ci intake/yr per Ci deposited/m^2-yr) is numerically equal to the ratio of the total integrated intake to the acute surface deposition (Ci intake per Ci deposited/m^2). Thus, if we apply equation C-1 for equilibrium conditions (long buildup times), it is appropriate to use the equation to compute values of RI_{np} for our analysis. The expressions for C_n^P and C_n^L are basically the same as given in equation 49, page 40, of AIRDOS-EPA (Mo79) modified to account for removal of radionuclides from the plant root zone to a soil sink (λ_{sn}). The equations are

$$C_n^P = d_n \left[\frac{DDI\ f_{cp}\ [\ 1 - e^{-\lambda_{En} t_{ep}}\ ]}{Y_{vp} \lambda_{En}} + \frac{B_{iv2}\ [\ 1 - e^{-(\lambda_{Dn} + \lambda_{Sn}) t_b}\ ]}{P\ (\lambda_{Dn} + \lambda_{Sn})} \right] e^{-\lambda_{Dn} t_{hp}} \tag{C-2}$$

and

$$C_n^L = d_n \left[\frac{DDI\ f_{cL}\ [\ 1 - e^{-\lambda_{En} t_{eL}}\ ]}{Y_{vL} \lambda_{En}} + \frac{B_{iv1}\ [\ 1 - e^{-(\lambda_{Dn} + \lambda_{Sn}) t_b}\ ]}{P\ (\lambda_{Dn} + \lambda_{Sn})} \right] e^{-\lambda_{Dn} t_{hL}} \tag{C-3}$$

where

DDI = fraction of nuclide retained on plant foliage which remains after washing (dimensionless),

f_{cp} and f_{cL} = fraction of deposited nuclide retained on edible portions of produce and leafy vegetables, respectively (dimensionless),

λ_{En} = the effective removal rate constant for nuclide n from crops (yr^{-1}),

t_{ep} and t_{eL} = time period that crops are exposed to contamination during growing season for produce and leafy vegetables, respectively (yr),

Y_{vp} and Y_{vL} = agricultural productivity (yield) of the edible portion of produce (kg wet/m^2) and leafy vegetables (kg dry/m^2),

B_{iv2} and B_{iv1} = the concentration factor for uptake of radionuclide n from soil by edible parts of crops for produce (Ci/kg wet plant per Ci/kg dry soil) and leafy vegetables (Ci/kg dry plant per Ci/kg dry soil), respectively,

λ_{Dn} = radioactive decay constant for nuclide n (yr^{-1}),

λ_{Sn} = removal rate constant for transfer of nuclide n from the root zone to the soil below the root zone (yr^{-1}),

t_b = period of long-term buildup of activity in soil (yr),

P = effective density of the root zone (top 15 cm of soil) (kg dry soil/square meter), and

t_{hp} and t_{hL} = a holdup time that represents the time interval between harvest and consumption of produce and leafy vegetables (yr).

An implicit assumption in equations C-2 and C-3 is that irrigation water deposits radionuclides on the ground surface continuously. This is a conservative assumption since irrigation would actually take place for less than the full year in many locations within the U.S.

As mentioned above, a significant change was made in equations C-2 and C-3 that is not included in the models in the AIRDOS-EPA program manual. The loss of radionuclides from the soil root zone due to leaching of radionuclides to the soil below the root zone was accounted for by adding the λ_{Sn} term to the equation. This loss mechanism can be important for long-lived radionuclides and this is the reason for including the modification in the equations. The addition of this loss mechanism causes the majority of the total intake of radionuclides from deposition on crop land to occur over a shorter time period after deposition of radioactivity in the environment than would be the case if radioactive decay were the only mechanism for removal from the soil root zone. Substituting equations C-2 and C-3 into C-1 yields

$$RI_{np} = U^{p}\left[\frac{DDI\ f_{cp}\ [\ 1 - e^{-\lambda_{En}t_{ep}}\ ]}{Y_{vp}\ \lambda_{En}} + \frac{B_{iv2}\ [\ 1 - e^{-(\lambda_{Dn}+\lambda_{Sn})t_{b}}\ ]}{P\ (\lambda_{Dn} + \lambda_{Sn})}\right] e^{-\lambda_{Dn}t_{hp}}$$

$$+\ U^{L}\left[\frac{DDI\ f_{cL}\ [\ 1 - e^{-\lambda_{En}t_{eL}}\ ]}{Y_{vL}\ \lambda_{En}} + \frac{B_{iv1}\ [\ 1 - e^{-(\lambda_{Dn}+\lambda_{Sn})t_{b}}\ ]}{P\ (\lambda_{Dn} + \lambda_{Sn})}\right] e^{-\lambda_{Dn}t_{hL}}. \tag{C-4}$$

The methods used to derive algorithms for RI_{np} for milk and beef are similar to those used to derive equation C-1. For milk, we may write

$$RI_{np} = \frac{U^{m}\ C_{n}^{m}}{d_{n}} \tag{C-5}$$

where

U^{m} = quantity of milk consumed by a human receptor per unit time (liters/yr),

C_{n}^{m} = concentration of nuclide n in milk (Ci/liters), and d_{n} is as defined previously. The algorithm for C_{n}^{m} is obtained using the methodology discussed on pages 42 and 43 of AIRDOS-EPA (Mo79) and can be written as

$$C_{n}^{m} = F_{m}\ Q_{m}\ e^{-\lambda_{Dn}t_{fm}}\left\{\begin{matrix}\dfrac{f_{cf}\ \left[1-e^{-\lambda_{En}t_{em}}\right]}{Y_{vf}\ \ \lambda_{En}} \\ \\ +\ \dfrac{B_{iv1}\left[1-e^{-(\lambda_{Dn}+\ \lambda_{Sn})t_{b}}\right]}{P(\lambda_{Dn}+\lambda_{Sn})}\end{matrix}\right\}\left[f_{p}f_{s} + (1-f_{p}f_{s})e^{-\lambda_{Dn}t_{hff}}\right]d_{n} \tag{C-6}$$

where

F_m = average fraction of a cow's daily intake of radionuclide n which appears in each liter of milk (Ci/liter per Ci/day intake),

Q_m = amount of feed consumed by the cow (kg dry/day),

t_{fm} = average transport time of the activity from the feed into the milk and to the receptor (yr),

f_{cf} = fraction of the deposited radionuclides retained on edible portions of animal feed crops (dimensionless),

t_{em} = time period that milk cow feed crops are exposed to contamination during the growing season (yr),

Y_{vf} = agricultural productivity (yield) of the edible portion of animal feed crops (kg dry/m^2),

f_p = fraction of year that animals graze on pasture (dimensionless),

f_s = fraction of feed that is pasture grass when the animals graze on pasture (dimensionless),

t_{hsf} = the time interval between harvest and consumption for stored animal feed (yr),

and the other terms are as defined previously. Substituting equation C-6 into equation C-5 yields

$$RI_{np} = U^m F_m Q_m \, e^{-\lambda_{Dn} t_{fm}} \left\{ \frac{f_{cf} \left[1-e^{-\lambda_{En} t_{em}}\right]}{Y_{vf} \; \lambda_{En}} + \frac{B_{ivl} \left[1-e^{-(\lambda_{Dn}+\lambda_{Sn}) t_b}\right]}{P(\lambda_{Dn}+\lambda_{Sn})} \right\} \left[f_p \, f_s + (1-f_p \, f_s) \, e^{-\lambda_{Dn} t_{hsf}}\right] \tag{C-7}$$

For beef, RI_{np} is determined by

$$RI_{np} = \frac{U^F \, C_n^F}{d_n} \tag{C-8}$$

where

U^F = quantity of meat consumed by a human receptor per unit time (kg/yr)

C_n^F = concentration of radionuclide n in meat (Ci/kg)

and d_n is as defined previously. The algorithm used to determine values for C_n^F is obtained as described on pages 43 and 44 of AIRDOS-EPA (Mo79) and is

$$C_n^F = F_f \, Q_f \, e^{-\lambda_{Dn} t_s} \left\{ \frac{f_{cf} \left[1-e^{-\lambda_{En} t_{ef}}\right]}{Y_{vf} \; \lambda_{En}} + \frac{B_{ivl} \left[1-e^{-(\lambda_{Dn}+\lambda_{Sn}) t_b}\right]}{P(\lambda_{Dn}+\lambda_{Sn})} \right\} \left[f_p f_s + (1-f_p f_s) \, e^{-\lambda_{Dn} t_{hsf}}\right] d_n \tag{C-9}$$

where

F_f = average fraction of a cow's daily intake of radionuclide n which appears in each kilogram of meat (Ci/kg per Ci/day intake),

Q_f = amount of feed consumed by the cow (kg dry/day),

t_s = average time from slaughter to consumption (yr),

t_{ef} = time period that meat animal feed crops are exposed to contamination during the growing season (yr),

and the other terms are as defined previously. Substituting equation C-9 into equation C-8 yields

$$RI_{np} = U^f F_f Q_f \, e^{-\lambda_{Dn} t_s} \left\{ \frac{f_{cf} \left[1-e^{-\lambda_{En} t_{ef}} \right]}{Y_{vf} \; \lambda_{En}} + \frac{B_{ivl} \left[1-e^{-(\lambda_{Dn} + \lambda_{Sn}) t_b} \right]}{P(\lambda_{Dn} + \lambda_{Sn})} \right\} \left[f_p \, f_s + (1-f_p \, f_s) \, e^{-\lambda_{Dn} t_{hsf}} \right] \tag{C-10}$$

The parameter values chosen for this analysis are discussed below and the references used to derive these values are listed.

For Vegetative Food Crops

U^p = 176 kg wet vegetation/yr (Mo79)

DD1 = 1.0 (conservative assumption)

f_{cp} = 0.052 (Ba84)

λ_{En} = 18.4 yr^{-1} (all nuclides except I) (Mo79)

This is based on a weathering half time of 13.75 d.

λ_{En} = 31.6 yr^{-1} (I isotopes) (Ba84)

This is based on a weathering half time of 8 d.

$t_{ep} = t_{eL}$ = 100 d = 0.274 yr (Ba83a)

Y_{vp} = 1.6 kg wet/m^2 (Ba84)

The parameters λ_{Dn} and λ_{Sn} are discussed in Chapter 5 and values for various radionuclides are given in Tables 5-1 and 5-4. Values for λ_{Sn} are also repeated in Table C-1 of this appendix. As discussed earlier, equation C-4, C-7, and C-10 must be evaluated at equilibrium so the term $e^{-(\lambda_{Dn} + \lambda_{Sn})t_b}$ must drop out of the equation and a value for t_b is not needed.

P = 215 kg dry soil/m^2 (Mo79)

$t_{hp} = t_{hL}$ = 336 hr (Mo79) (produce and leafy vegetables)

For all radionuclides considered, the half lifes are long enough such that $e^{-\lambda_{Dn} t_{hp}}$ and $e^{-\lambda_{Dn} t_{hL}}$ are ~1.0 so that these terms can be set equal to 1 for our analysis.

U_L = 1.2 kg dry veg./yr (Mo79, Ba83a)

f_{cL} = 0.15 (Ba84)

Y_{vL} = 0.12 kg dry veg./m^2 (Ba84)

The values for B_{iv1} and B_{iv2} are radionuclide specific and are listed in Table C-1. The values are based on information provided by Baes (Ba84) except where noted in the table.

For Milk

U^m = 112 liters milk/yr (Mo79)

F_m values are specific to each radionuclide and are listed in Table C-1. The values are based on information provided by Baes (Ba84) except where noted otherwise in the table.

Q_m = 18.1 kg dry veg./day (Sh82)

t_{fm} = 4 days = 0.011 yr (Mo79)

TABLE C-1: Radionuclide specific data used to compute RI_{np}

Nuclide	B_{iv1} $\frac{\text{pCi/kg dry plant}}{\text{pCi/kg dry soil}}$	B_{iv2} $\frac{\text{pCi/kg wet plant}}{\text{pCi/kg dry soil}}$	λ_{Sn} (yr^{-1}) +	F_m $\frac{\text{pCi/l}}{\text{pCi/day intake}}$	F_f $\frac{\text{pCi/kg}}{\text{pCi/day intake}}$	K_d ml/g
C	NA	NA	NA	NA	NA	NA
Ni	6.00 E-2	2.60 E-2	5.40 E-3	1.00 E-3	2.00 E-3***	150
Sr	2.50 E0	1.10 E-1	2.31 E-2	1.50 E-3	3.00 E-4	35
Zr	7.20 E-2**	7.70 E-4**	2.70 E-4	3.00 E-5	2.00 E-2***	3,000
Tc	9.50 E0	6.40 E-1	4.90 E-1	1.00 E-2	8.50 E-3	1.5
Sn	3.00 E-2	2.60 E-3	3.24 E-3	1.00 E-3	8.00 E-2	250
I	1.50 E-1	2.10 E-2	1.57 E-3	1.00 E-2	7.00 E-3	10
Cs	8.00 E-2	1.30 E-2	8.10 E-4	7.00 E-3	2.00 E-2	1,000
Sm	1.00 E-2	1.70 E-3	1.25 E-3	2.00 E-5	5.00 E-3	650
Pb	4.50 E-2	3.90 E-3	9.00 E-4	2.50 E-4	3.00 E-4	900
Ra	1.50 E-2	6.40 E-4	1.80 E-3	4.50 E-4	5.80 E-4*	450
Ac	3.50 E-3	1.50 E-4	5.40 E-4	2.00 E-5	2.50 E-5	1,500
Th	8.50 E-4	3.60 E-5	5.40 E-6	5.00 E-6	6.00 E-6	150,000
Pa	2.50 E-3	1.10 E-4	3.24 E-4	5.00 E-6	1.00 E-5	2,500
U	8.50 E-3	1.70 E-3	1.80 E-3	6.00 E-4	2.00 E-4	450
Np	1.00 E-1	4.30 E-3	2.69 E-2	1.00 E-5$^{\alpha}$	2.00 E-4$^{\alpha\alpha}$	30
Pu	4.50 E-4	1.90 E-5	1.80 E-4	1.00 E-7	2.00 E-6***	4,500
Am	5.50 E-3	1.10 E-4	1.16 E-3	4.00 E-7	3.50 E-6	700
Cm	8.50 E-4	6.40 E-6	4.05 E-4	2.00 E-5	3.50 E-6	2,000

* Value is arithmetic average of values given by Baes (Ba84) and Ng (Ng82a).

** Value is based on data suggested by Ng (Ng82b).

*** Value is based on data suggested by Ng (Ng82a).

α Value based on data suggested by Schaffer (Sc83).

αα Value based on judgement after reviewing data from Baes (Ba84), Ng (Ng82a), and Schaffer (Sc83).

+ See Section C.6 of this appendix for explanation of methodology used to compute λ_{Sn}.

f_{cf} = 0.57 (Mo79, Ba83a)

t_{em} = 65 days = 0.178 yr (Ba83a, Sh82)

Y_{vf} = 0.28 kg dry veg./m^2

t_{hsf} = 90 days = 0.246 yr (Mo79) (stored feed)

For the radionuclides in this analysis, the half lifes are long enough such that $e^{-\lambda_{Dn} t_{hsf}}$ are ~1. This makes it unnecessary to select values for f_p and f_s since they drop out of the equation. Values for the other parameters in equation C-7 are the same as those used for the vegetative food crops.

For Meat

U^F = 94 kg meat/yr (Mo79)

F_f values are specific to each radionuclide and are listed in Table C-1. The values are based on information provided by Baes (Ba84) except where noted.

Q_f = 8.3 kg dry veg./day (Sh82)

t_s = 20 days = 0.055 yr (Mo79)

t_{ef} = 65 days = 0.178 yr (Ba83a, Sh82)

Values for the other parameters in equation C-10 are the same as those used for the milk pathway.

Using the algorithms and parameter values discussed in this section, the values for RI_{np} for vegetative food crops, milk and meat can be derived. These values are listed in Table 5-3.

C.2 Persons Fed per Unit Area (CP_p)

The parameter CP_p expresses the number of persons who can be fed per unit area of land for the vegetative food crops, milk and meat pathways. The deviation of this parameter for the various foods will be discussed in this section.

Vegetative Food Crops

Shor (Sh82) gives agricultural productivity for four different categories of vegetative food crops. In a recent report, Baes (Ba84) lists estimates of the relative importance of these categories in the human diet. This data is recorded in Table C-2.

TABLE C-2: Productivity and relative importance of vegetative human food crops

Crop Category	Productivity kg wet/m^2	Percent of Vegetative Human Food
Leafy vegetables	2.44	5.8
Exposed produce	1.65	20.0
Protected produce	0.91	42.2
Grains	0.23	32.0

We have assumed that one food crop per year is raised and that each person consumes 194 kg wet of vegetative food crops per year (Mo79) so that CP_p (veg) may be calculated as

$$CP_p\ (\text{veg}) = \frac{2.44(.058) + 1.65(.20) + 0.91(.422) + 0.23(.32)\ \dfrac{\text{kg wet}}{\text{m}^2\text{-yr}}}{194\ \dfrac{\text{kg wet}}{\text{yr-person}}}$$

$$CP_p\ (\text{veg}) = 4.79\ \text{E-3 persons fed/m}^2$$

Milk

Shor (Sh82) gives agricultural productivity and areal yield data for four categories of cattle feed crops. She also discussed the fraction of total cattle diet furnished by each feed crop. This data is listed in Table C-3.

TABLE C-3: Areal yield and fraction of diet for cattle feed crops

Crop Category	Areal Yield (kg dry/m^2-yr)	Fraction of Cattle Feed
Pasture	0.09	0.55
Grain	0.31*	0.17
Hay	0.46	0.21
Silage	0.66*	0.07

* Productivity (kg dry/m^2); assume 1 crop/yr to obtain areal yield.

Shor (Sh82) also provides the following data:

Milk Cow Feed Rate $= Q_m = 18.1$ kg dry veg./day consumed by milk cows

Milk Production $= 12.9\ \frac{\text{liters}}{\text{day-cow}}$

From the AIRDOS-EPA computer code manual (Mo79)

Milk consumption by man $= 112\ \frac{\text{liters}}{\text{yr-man}}$

Then, using the data from Table C-3 and the above listed values,

$$CP_p(\text{milk}) = \frac{\left[.09(.55)+.31(.17)+(.46)(.21)+.66(.07)\ \frac{\text{kg dry veg}}{\text{yr-m}^2}\right]\left(12.9\ \frac{\text{liters milk}}{\text{day cow}}\right)}{\left(18.1\ \frac{\text{kg dry veg}}{\text{day-cow}}\right)\left(112\ \frac{\text{liters milk}}{\text{y-man}}\right)}$$

$$CP_p(\text{milk}) = 1.56\ \text{E-3}\ \frac{\text{man fed}}{\text{m}^2}$$

Meat

The data on areal yield and percentage of cattle feed listed in Table C-3 were utilized in deriving CP_p for the meat pathway. Other data listed by Shor (Sh82) which were used are:

$Q_f = 8.3\ \frac{\text{kg dry feed}}{\text{day}}$

Slaughter factor = 0.34/yr

Live weight at slaughter $= 477\ \frac{\text{kg}}{\text{animal}}$

$$\frac{\text{dressed weight}}{\text{live weight}} = 0.56$$

Therefore,

$$\text{Meat production} = \left(477\ \frac{\text{kg live}}{\text{animal}}\right)\left(0.56\ \frac{\text{kg meat}}{\text{kg live}}\right)\left(\frac{0.34}{\text{y}}\right)$$

$$= 90.8\ \frac{\text{kg meat}}{\text{animal-yr}} = 0.25\ \frac{\text{kg meat}}{\text{animal-day}}$$

From the AIRDOS-EPA computer program manual, human annual meat consumption is 94 kg meat/yr. Using this data, we can derive the CP_p for meat as

$$CP_p(\text{meat}) = \frac{\left[.09(.55)+.31(.17)+(.46)(.21)+.66(.07)\ \frac{\text{kg dry veg}}{\text{yr-m}^2}\right]\left(0.25\ \frac{\text{kg meat}}{\text{animal-day}}\right)}{\left(8.3\ \frac{\text{kg dry veg}}{\text{day-cow}}\right)\left(94\ \frac{\text{kg meat}}{\text{y-person}}\right)}$$

$$CP_p(\text{meat}) = 7.85\text{ E-5}\ \frac{\text{man fed}}{\text{m}^2}\ .$$

In summary, the values used for CP_p in our analysis are listed in Table C-4.

TABLE C-4: Values for persons fed per unit area of land (CP_p)

Food	CP_p person fed/m^2
Vegetative Food Crops	4.79 E-3
Milk	1.56 E-3
Meat	7.85 E-5

C.3 Fraction of River Flow Used for Irrigation (f_R)

The fraction of the river flow used for irrigation, f_R, was set equal to 0.1 in this analysis. This number is representative of the average fraction of total surface water flow used for irrigation for the U.S. The reader may obtain a feel for the variation in f_R for various regions of the U.S. by referring to the data in Table C-5.

TABLE C-5: Fraction of river flow used for irrigation (f_R) by Water Resources Council region for 1975 (Mu77)

Water Resources Council Region	f_R
New England	0.001
Mid-Atlantic	0.001
South Atlantic-Gulf	0.009
Great Lakes	0.001
Ohio	0
Tennessee	0
Upper Mississippi	0.001
Lower Mississippi	0.020
Souris-Red-Rainy	0.003
Missouri Basin	0.370
Arkansas-White-Red	0.029
Texas-Gulf	0.031
Rio Grande	0.580
Upper Colorado	0.285
Lower Colorado	0.969
Great Basin	0.667
Pacific Northwest	0.114
California	0.307
U.S. (conterminous)	0.070

For the conterminous U.S., the f_R values was rounded from 0.07 to 0.1. The data for the conterminous U.S. from the above table have been compared to data from other references which generally support the f_R value of 0.1 (WIC70, WRC78).

C.4 Population Density (PD_p)

The value used for population density is 6.67 E-5 persons/m^2. This value is the world average population density and was obtained by dividing the assumed population of 10^{10} persons* by the land surface area of the earth of 1.5 E14 m^2 (Wo79). A review of the data listed in Table C-6 will show that the PD_p used is within the range of current values for various regions of the U.S. As the world population increases, the U.S. population would also increase.

TABLE C-6: U.S. regional values for population density (PD_p) (USDC83)

Region	Population Density for 1982 (persons/m^2)
U.S. Average	2.51 E-5
New England	7.65 E-5
Middle Atlantic	1.43 E-4
East North Central	6.57 E-5
West North Central	1.31 E-5
South Atlantic	5.52 E-5
East South Central	3.21 E-5
West South Central	2.28 E-5
Mountain	5.41 E-6
Pacific	1.43 E-5

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*The current world population is about 3.8 E9. However, an estimate of average world population during the time period involved in this calculation is 10^{10} people (UN77).

C.5 Correction Factor for the Ground Surface Risk Factors (GC_{np})

The risk conversion factors used for radionuclides deposited on the ground were derived using the assumption that the material remains on the surface. Over the long time periods involved in these calculations, the radionuclides will move vertically downward into the soil. The gamma radiation emitted from these nuclides is partially shielded by the soil such that the dose equivalent per unit deposition is less than given by the surface dose conversion factors. We derived correction factors for each nuclide by assuming that the radioactivity was uniformly distributed vertically within the soil root zone (see the discussion in Section 3.1.6). The algorithm used for calculating the correction factor is

$$GC_{np} = \frac{\sum_i f_i \left[D_{isl}(\gamma) \right]}{\sum_i f_i \left[15\ D_{ipl}(\gamma) \right]} \qquad \text{(C-11)}$$

where

GC_{np} = radionuclide specific factor used to correct ground surface fatal cancer risk factors to account for uniform distribution of radionuclides within the 15 cm soil root zone (dimensionless),

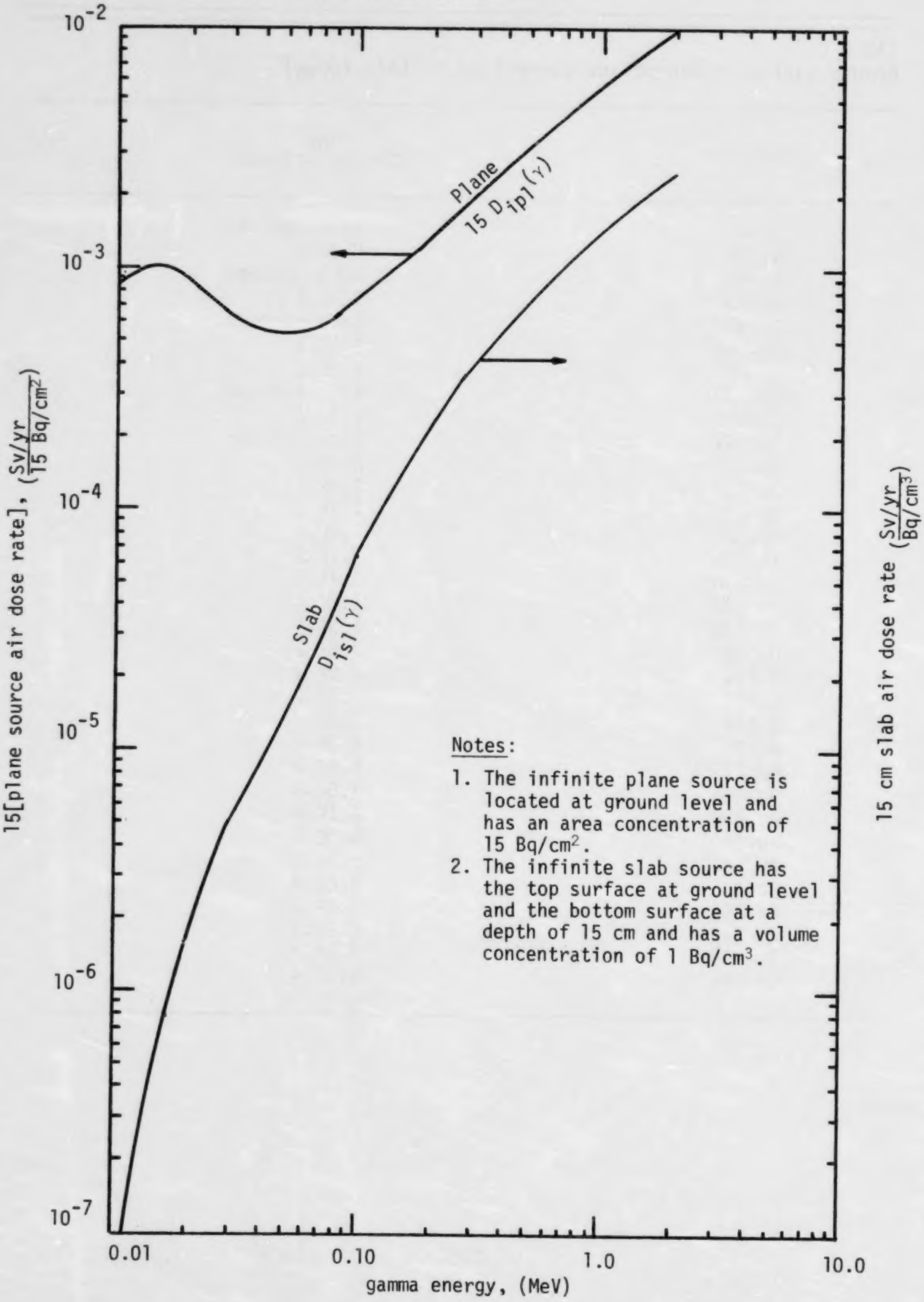
f_i = gamma intensity for the $i^{\underline{th}}$ gamma (gammas emitted/disintegration),

$D_{isl}(\gamma)$ = dose rate in air due to 1 Bq/cm^3 ground volume concentration within the 15 cm soil root zone for gamma energy i (Sv/y),

$D_{ipl}(\gamma)$ = dose rate in air due to 1 Bq/cm^2 ground surface concentration for gamma energy i (Sv/y).

The summations are for all gammas emitted by the parent radionuclide and the daughter radionuclides which were included in deriving the risk conversion factors. The multiplier, 15, in the denominator of the equation is to adjust the radionuclide content of the soil surface to equal that of the 15 cm slab representing the soil root zone. Values for $D_{isl}(\gamma)$ and $D_{ipl}(\gamma)$, as a function of gamma energy, were furnished by Kocher (Ko83b). $D_{isl}(\gamma)$ and 15 $D_{ipl}(\gamma)$ are plotted as a function of gamma energy in Figure C-1. Using the algorithm given above, the gamma energy intensity information for the parents and significant daughters given by Kocher (Ko81a) and the data plotted in Figure C-1, the radionuclide specific correction factors listed in Table C-7 were computed. These factors were multiplied by the RADRISK risk conversion factors (derived assuming surface deposition) to obtain risk conversion factors for uniform distribution of radionuclides within the 15 cm soil root zone.

Figure C-1. Dose rates in air at 1 meter above ground for an infinite uniform plane source and an infinite uniform slab source



Notes:

1. The infinite plane source is located at ground level and has an area concentration of 15 Bq/cm^2.
2. The infinite slab source has the top surface at ground level and the bottom surface at a depth of 15 cm and has a volume concentration of 1 Bq/cm^3.

TABLE C-7:
Ground surface radionuclide correction factors (GC_{np})

Nuclide	GC_{np} (Dimensionless)
C-14	Beta emitter
Ni-59	8.8E-5
Sr-90	Beta emitter
Zr-93	1.3E-3
Tc-99	Beta emitter
Sn-126	2.2E-1
I-129	1.0E-2
Cs-135	Beta emitter
Cs-137	2.4E-1
Sm-151	Beta emitter
Pb-210	2.3E-2
Ra-226	2.4E-1
Ra-228	1.8E-1
Ac-227	1.5E-1
Th-229	9.7E-2
Th-230	2.4E-1
Th-232	1.8E-1
Pa-231	1.3E-1
U-233	7.5E-2
U-234	2.9E-2
U-235	7.3E-2
U-236	2.5E-4
U-238	4.3E-2
Np-237	9.2E-2
Pu-238	3.8E-4
Pu-239	4.1E-4
Pu-240	4.0E-4
Pu-241	1.2E-2
Pu-242	4.0E-4
Am-241	1.2E-2
Am-243	6.9E-2
Cm-245	3.3E-2
Cm-246	4.0E-4

C.6 Leaching Removal Rate Constant for Soil (λ_{Sn})

The leaching rate constant for transfer of nuclides from available to unavailable soil is λ_{Sn}. The value for λ_{Sn} is nuclide dependent and is determined by a method described by Baes (Ba79a). The algorithm is

$$\lambda_{Sn} = \frac{V_w}{d_s\left[1 + \frac{\rho}{\theta}\ K_d\right]} \tag{C-12}$$

where

λ_{Sn} = leaching removal rate constant from soil root zone (yr^{-1}),

V_w = velocity of vertical water percolation (cm/yr),

d_s = depth of soil root zone (cm),

ρ = soil bulk density (g/cm^3)

θ = soil water content (ml/cm^3)

K_d = equilibrium distribution coefficient of the nuclide species between soil and water (ml/g)

Parameter values for these generic calculations were chosen after discussion with C.F. Baes III of Oak Ridge National Laboratory. The parameter values and references are listed below:

V_w and ρ - used median value from Table 3.19, NUREG/CR-1004 (Ba79a)

$V_w = 74$ cm/yr

$\rho = 1.4$ g/cm^3

$d_s = 15$ cm (assumed soil root zone depth)

Θ - used average of median field capacity volumetric water content and median wilting point volumetric water content for all soils from Table 2 of Baes and Sharp 1983 article in the Journal of Environmental Quality (Ba83b)

$$\Theta = 0.23 \frac{\text{ml}}{\text{cm}^3}$$

K_d - values used are the default values recommended by Baes in ORNL-5786 (Ba84)

K_d values are nuclide specific and are listed in Table C-1.

Using the above listed parameter values, the values for λ_{Sn} listed in Table C-1 were computed, except for I-129. For I-129, data from Kocher's dynamic model of the global iodine cycle (Ko81b, Ko83b) were applied to determine λ_{Sn}. Kocher has a surface soil region with a depth of 100 cm in his model. Dr. Kocher recommended that we consider transfer from his surface soil region to the ocean mixed layer, the shallow land subsurface region and the deep land subsurface region in determining a value for λ_{Sn}. The rate constants listed by Kocher for transfer from his 100 cm deep surface soil region to the other three regions are listed in Table C-8. We linearly scaled these rate constants to obtain rate constants for our 15 cm soil root zone (Table C-8). The summation of these three rate constants gives a value for λ_{Sn} for I-129 of 1.57 E-3 yr^{-1}.

TABLE C-8:
Determination of λ_{Sn} for I-129

Transfer from Surface Soil Region to	Rate Constant, (yr^{-1})	
	100 cm depth	15 cm depth
Ocean Mixed Layer	2.0 E-4	1.3 E-3
Shallow Subsurface Region	3.5 E-5	2.3 E-4
Deep Subsurface Region	7.0 E-7	4.7 E-6
	$\sum$ =	1.57 E-3 yr^{-1}

C.7 Ratio of Persons Drinking Water and Eating Fish to River Flow Rate

P_R is the number of people who drink water and/or eat fish from a river and R is the river flow rate. The ratio P_R/R is needed in several equations, and it can be determined for the purpose of this generic evaluation without obtaining site specific data. Utilizing data from Annex D of the 1977 UNSCEAR Report (UN77), the annual flow rate of the rivers of the world is 3E+16 liters/yr. If one assumes a constant world population of 10^{10} persons*, the ratio P_R/R is 3.3E-7 person-yr/liter. This value for the ratio P_R/R is midrange of values found for various river basins in the United States. These values ranged from a high of 5.73E-7 for the Lower Colorado Water Resources Council region to a low of 2.39E-8 for the Pacific Northwest Water Resources Council region, based on 1975 river flow and population estimates. Table C-9 lists these ratios for the various Water Resources Council regions.

C.8 Total Fraction of Initial River Inventory Deposited to Cropland for Radionuclides Rapidly Transferred Through Soil (Radionuclide Recycle)

The algorithms used to compute environmental risk commitment from irrigated food crop consumption do not include the consideration of recycle of radionuclides deposited on the cropland back to the river and a

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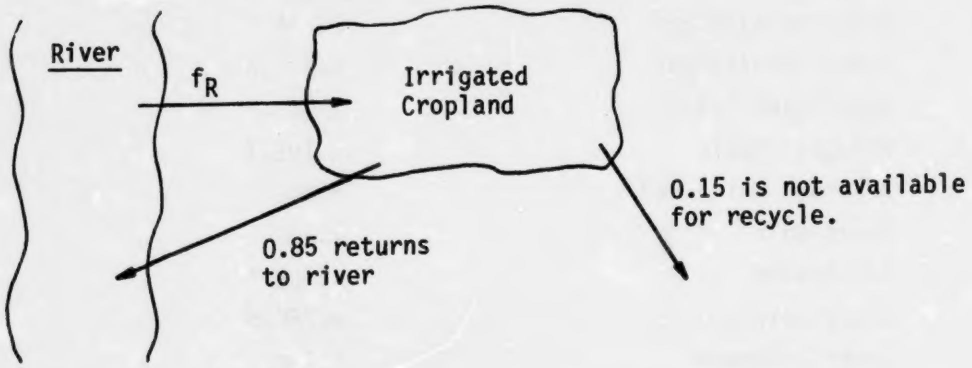
*The current world population is about 3.8E9. However, an estimate of average world population during the time period involved in this calculation is 10^{10} people (UN77).

TABLE C-9: Ratio of persons drinking water and eating fish to river flow rate by Water Resources Council region (Mu77, USDC83)

Water Resources Council Region	P_R/R (Person-yr/liter)
New England	1.32E-7
Mid-Atlantic	2.88E-7
South Atlantic-Gulf	1.03E-7
Great Lakes	2.02E-7
Ohio	1.50E-7
Tennessee	5.62E-8
Upper Mississippi	2.00E-7
Lower Mississippi	6.27E-8
Souris-Red-Rainy	1.78E-7
Missouri Basin	1.19E-7
Arkansas-White-Red	9.24E-8
Texas-Gulf	1.90E-7
Rio Grande	4.40E-7
Upper Colorado	8.76E-8
Lower Colorado	5.73E-7
Great Basin	1.09E-7
Pacific Northwest	2.39E-8
California	2.52E-7
U.S. (conterminous)	1.30E-7

redeposition of this material back to the cropland via irrigation. We assume that when the radionuclides are removed from the soil root zone (by leaching) they are no longer available for uptake by foodcrops. For radionuclides which leach rapidly through the soil, it is of interest to consider the effects of recycling. Of the radionuclides we consider, the one most subject to this phenomenon is Tc-99.

Dr. Kocher (Ko83a) has suggested a bounding calculation to determine the importance of recycle in making material available for plant uptake from soil. The computation is shown below.



Assumptions:

- Neglect radioactive decay as a removal mechanism.
- 85 percent of radionuclide deposited on cropland immediately recycles back to the river (λ_{Sn} is very large).
- 15 percent of radionuclide deposited on cropland is permanently fixed within the soil below the plant-root-zone and is no longer available for uptake or recycle.

Then let

f_R = fraction of river flow used for irrigation.
I_o = initial inventory in river (Ci).
I_F = final inventory on cropland (Ci).

We may write

$$I_F = f_R\ I_o + (0.85 f_R I_o) f_R + (0.85 f_R^2 I_o)(0.85)(f_R) \qquad \text{(C-13)}$$
$$+ [(0.85)^2\ f_R^3\ I_o](0.85)(f_R) + [(0.85)^3\ f_R^4\ I_o](0.85)(f_R) + \ . \ . \ .$$

Then

$$\frac{I_f}{I_o} = f_R + (0.85 f_R) f_R + (0.85 f_R)^2 f_R + (0.85 f_R)^3 f_R + (0.85 f_R)^4 f_R + \ . \ . \ .$$

The above series is a geometric progression where the square of the common ratio $(0.85 f_R)$ is less than one. For this case, the value of I_f/I_o for an infinite summation is

$$\frac{I_f}{I_o} = \frac{f_R}{1-0.85 f_R} \qquad \text{(Bu48)} \qquad \text{(C-14)}$$

The ratio I_f/I_o represents the total fraction of the initial inventory in the river which is deposited on cropland due to infinite recycling of radionuclides. For our chosen value of f_R of 0.10, we have

$$\frac{I_f}{I_o} = \frac{0.10}{1-0.85(0.10)} \simeq 0.11.$$

Since this ratio is very close to our chosen value of f_R of 0.10, the effects of recycling are not significant in our calculations.

C.9 Tabulation of Estimated Range of Parameter Values

A specific request of the Science Advisory Board High-Level Radioactive Waste Disposal Subcommittee was that we include, in the final pathways report, a listing of parameter values used in the analysis and an estimate, where possible, of the range of values for the parameters. This information is included in Table C-10. In addition to listing the estimated range of parameter values, the values used for our analyses, which are also listed in other sections of this report, are restated in the table. The parameters which are essentially invarient (such as earth land area) are not listed in the table.

For some parameters, a range of values was not given in the references we consulted and only the default value which we used is given. For others, intuition dictates that the range given appears to be too narrow (for example, the range of K_d for Sm, 600-650 ml/g). If more data were available, it is probable that the range would be larger. In some cases, we applied a parameter value for our analysis which was near an extreme of the range rather than one near the midpoint. This is an indication that the bulk of the data had values near this extreme and/or that we had more confidence in the data near this extreme.

TABLE C-10:
Listing of parameter value ranges

Parameter	Estimated Range of Values	Value Used	Notes
λ_L	10^{-6} to 10^{-2} per yr	10^{-4}/yr	
P_R/R and P_{FF}/R	2.4E-8 to 5.7E-7 (Pacific Northwest)(Lower Colorado)	3.3E-7	Estimated range for USA based on 1975 data (Mu77, USDC83). Value used is world average number for a stable world population which is about 2.6 times the current population (UN77).
I_w	320 to 712	603 l/y (Ref. man value)	Range is measured data. Ref. man value is set higher than most measured values. See pages 358 and 360 of ICRP No. 23 (ICRP75).
I_f		1.0 kg/y (fresh water fish)	Range is per caput. Data from page 349, ICRP No. 23 (ICRP75). Values used from UNSCEAR (UN77).
I_p	4.4 to 13.9 (Supplies of fish)	6.0 kg/y (Ocean fish)	
		1.0 kg/y (Ocean shellfish)	
f_p	Values could vary from 0 to 1.0	0.50 (food crops)	Value used column lists the fraction of crop land used for the various food crops.
		0.25 (Milk)	
		0.25 (Meat)	
f_R	0 to 0.97	0.10	Value used is U.S. average (See Table C-5).

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used	Notes
CP_p	5.1E-5 to 9.3E-2	4.79E-3 (Food crops) $\frac{\text{man fed}}{m^2}$	(Ba79b, Ho82b, USDA82, Ba84)
	2.1E-5 to 1.4E-1	1.56E-3 (Milk) $\frac{\text{man fed}}{m^2}$	
	1.1E-6 to 1.0E-2	7.85E-5 (Meat) $\frac{\text{man fed}}{m^2}$	
PD_p	5.4E-6 (Mountain) to 1.4E-4 (Mid Atlantic)	6.67E-5 $\frac{\text{persons}}{m^2}$	Estimated range for USA based on 1982 data (USDC83). Value used is world average number for a stable world population which is about 2.6 times the current population (UN77, Wo79).
RF	1E-10 to 1E-8	$1E\text{-}9m^{-1}$	Weathered material. (Ri83, Be76).
λ_R	See Note	$1E\text{-}11sec^{-1}$	Calculated from range of RF using the equation $RF=\lambda_R/vg$ with vg=0.01 m/sec.
I_B	750 to 14,600	8400 $\frac{m^3}{y}$	(ICRP75) Minimum is for 1 yr old resting 24 hr/day. Maximum is for adult man doing heavy work 10 hr/day, light activity 6 hr/day and resting 8 hr/day. Value used is for Reference Man (Adult man).
f_{wt}	0.2 to 1.0	1.0	Estimated range is for various nuclides. Data from (NRC78, F171, De75).

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used	Notes
f_s	0.10 to 0.90	0.65	Value used is U.S. average. Range represents variation within U.S. (Mu77).
h_A	7,600m to 18,000m	13,000m	Value used is mean of estimated range. Estimated range from (Wo79).
v_{gn}	0.0004 to 0.09	0.01	Estimated range is from data in Meteorology and Atomic Energy, 1968 (AEC68).
v_{wn}	0.009 to 0.057	0.02	Estimated range is from data in Meteorology and Atomic Energy, 1968 (AEC68). There is not nearly so much data for v_{wn} as for v_{gn}. The available data suggests that v_{wn} is at least twice v_{gn}.
λ_{wn}		$48.7y^{-1}$	Value calculated from v_{wn} and h_A.

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used	Notes
CF_{np}* (Freshwater Fish)			
C	4550-5000	4550	(Th72)
Ni	100	100	(Th72)
Sr	7.0E-1 to 2.0E2	11	(Bl82b, Ri83)
Zr	3.3	3.3	(Th72)
Tc	11 to 75	43	(Bl82a)
Sn	100-10,000	3000	(Ri83, Th72)
I	10-132	33	(Ho79)
Cs	40-14,000	1300	(Pe83, Bl82b, Ho79)
Sm	25	25	(Th72)
Pb	100-300	100	(Th72)
Ra	3-750	50	(Bl82b, Pe83, Th72)
Ac	25	25	(Th72)
Th	30-80	30	(Th72, Pe83, Th72)
Pa	11	11	(Th72)
U	2-20	10	(Th72, Bl82b, Th72)
Np	10-10,000	500	(Th72, Ri83) Value of 10,000 was top of range used by Envirosphere Co. This was not a measured value.
Pu	0.1-50,000	8	(Bl82b, Ri83) Value of 50,000 appears to be an outlier. Most reported values are between 3.5 and 50.
Am	15 to 452	81	(Ri83)
Cm	25	25	(Th72)

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*CF_{np} data for ocean fish and ocean shellfish not included in this tabulation because of minor significance of these pathways.

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used	Notes
Parameters used in calculating λ_{Sn} (See equation C-12).			
V_w	36.5-376	74 cm/y	(Ba79a)
ρ	0.93-1.84	1.4 g/cm^3	(Ba79a)
θ	0.03-0.40	0.23 ml/cm^3	(Ba79a)
d_s		15 cm	(Ba79a)
K_d values (ml/g)			
C	NA	NA	
Ni	80-150	150	
Sr	0.15-3,300*	35	
Zr	2000-3000	3000	
Tc	0.0029-1.5*	1.5	
Sn	250	250	
I	0-10	10	
Cs	10-52,000*	1000	
Sm	600-650	650	
Pb	4.5-7640*	900	
Ra	100-450	450	
Ac	1000-1500	1500	
Th	2000-510,000*	150,000	
Pa	2500-4000	2500	
U	10.5-4400*	450	
Np	0.16-929*	30	
Pu	11-300,000*	4500	
Am	1.0-47,230*	700	
Cm	99.3-51,900*	2000	

*Range as listed in Table 2.13 (Ba84). Range estimates without * are based on default value in (Ba84) and value listed in Table C-3 of (Li77c). Values used are the default values in (Ba84).

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used	Notes
		Parameters used in calculating RI_{np}	
U^P	0 to 540	176 $\frac{\text{Kg wet}}{\text{y}}$	(Ho82b, Mo79)
DD1	0-1.0	1.0	
f_{cp}	0.027 to 0.073	0.052	(Ba84)
λ_w	1.7-128.0	18.4/y	(Ho82b, Mo79)
t_{ep}	60-180d	100d	(Ho82b, Ba84)
Y_{vp}	1.0E-2 to 5.6	1.6 $\frac{\text{kg wet}}{\text{m}^2}$	(Ba79b, Ba84)
P	100-300	215 $\frac{\text{kg dry}}{\text{m}^2}$	(Ho82b, Mo79)
t_{hp}	84-336	336 hr	(Bl77, Mo79)
U^L	0 to 3.7	1.2 kg dry/yr	(Ho82b, Ba84)
DD1	0-1.0	1.0	
f_{cL}	0.06-1.0	0.15	(Mi79, Ba84)
t_{eL}	40-120d	100d	(Ho82b, Ba84)
Y_{vL}	0.024 to 0.35	0.12 $\frac{\text{kg dry}}{\text{m}^2}$	(Ba79b, Ba84)
t_{hL}	84-336	336 hr	(Bl77, Mo79)

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used	Notes
U^m	0 to 600	112 l/yr	(Ho82b, Mo79)
Q_m	4 to 25	18.1 $\frac{\text{kg dry}}{\text{d}}$	(Ho82b, Sh82)
t_{fm}	3-4d	4d	(Bl77, Mo79)
f_{cf}	0.02 to 0.82	0.57	(Mi79, Mo79)
t_{em}	15-200d	65d	(Ho82b, Ba84)
Y_{vf}	0.03 to 1.7	0.28 $\frac{\text{kg dry}}{\text{m}^2}$	(Ba79b, Mo79)
t_{hsf}		90d	(Mo79)
U^f	0 to 300	94 kg/yr	(Ho82b, Mo79)
Q_f	1.6-18	8.3 kg dry/d	(Ho82b, Sh82)
t_s	12-20d	20d	(Bl77, Mo79)
t_{ef}	15-200d	65d	(Ho82b, Ba84)

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used*	Notes**
B_{ivl} (pCi/kg dry crop per pCi/kg dry soil)			
Ni	5.7E-3 to 5.5E-1	6.0E-2	(Ng82b)
Sr	4.3E-2 to 2.3E1	2.5	(Ba84, Ng82b)
Zr	7.2E-3 to 2.5E-1	7.0E-2	(Pe83, Ba84)
Tc	1.4 to 4.4E+1	9.5	(Ho80)
Sn	1.3E-2 to 3.0E-2	3.0E-2	(Ba84)
I	1.5E-2 to 2.0	1.5E-1	(Ng82b, Ba84)
Cs	3.8E-3 to 5.7E-1	8.0E-2	(Ng82b)
Sm	1.0E-2	1.0E-2	(Ba84)
Pb	1.3E-2 to 9.0E-1	4.5E-2	(Ba84)
Ra	2.4E-3 to 7.1E-1	1.5E-2	(Ba84)
Ac	3.5E-3 to 1.0E-2	3.5E-3	(Ba84, Mo79)
Th	8.5E-4 to 2.7E-3	8.5E-4	(Ba84, Mo79)
Pa	2.5E-3 to 1.0E-2	2.5E-3	(Ba84, Mo79)
U	8.5E-3	8.5E-3	(Ba84)
Np	4.8E-3 to 3.5	1.0E-1	(Pe83, Ng82b)
Pu	10^{-6} to 10^{-2}	4.5E-4	(Ba84)
Am	1.7E-3 to 5.5E-3	5.5E-3	(Pe83, Ba84)
Cm	3.0E-5 to 8.5E-4	8.5E-4	(Pe83, Ba84)

*Values used are default values given by Baes, et. al (Ba84) except for Zr which was taken from Ng, et. al (Ng82b).

**Notes indicate references used to estimate range of values.

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used*	Notes**
B_{iv2} (pCi/kg wet crop per pCi/kg dry soil)			
Ni	3.0E-4 to 1.5E-1	2.6E-2	(Ba84, Ng82b)
Sr	8.6E-5 to 1.1E-1	1.1E-1	(Ba84)
Zr	3.4E-6 to 1.8E-2	7.7E-4	(Ng82b)
Tc	9.6E-2 to 3.00***	6.4E-1	(Ba84)
Sn	1.0E-3 to 1.0E2	2.6E-3	(Ri83)
I	2.0E-4 to 1.5	2.1E-2	(Ng82b, Ng82c)
Cs	1.5E-5 to 6.8E-1	1.3E-2	(Ng82b, Ng82c)
Sm	1.7E-3 to 7.0E-3	1.7E-3	(Ba84)
Pb	3.0E-4 to 7.3E-2	3.9E-3	(Ng82c)
Ra	7.0E-5 to 7.5E-1	6.4E-4	(Ng82c)
Ac	1.5E-4 to 2.5E-3	1.5E-4	(Ba84, Mo79)
Th	3.0E-5 to 8.6E-3	3.6E-5	(Pe83, Ba84)
Pa	1.1E-4 to 2.5E-3	1.1E-4	(Ba84, Mo79)
U	3.0E-5 to 8.6E-3	1.7E-3	(Pe83, Ba84)
Np	5.7E-6 to 1.3E-1	4.3E-3	(Pe83, Ng82b)
Pu	3.8E-8 to 4.0E-2	1.9E-5	(Ng82c)
Am	2.3E-7 to 5.0E-3	1.1E-4	(Ng82c)
Cm	1.1E-6 to 1.2E-5	6.4E-6	(Pe83)

*Values used are default values given by Baes, et. al (Ba84) except for Zr which was taken from Ng, et. al (Ng82b).

**Notes indicate references used to estimate range of values.

***Range estimated from B_{iv1} range in Ho80 and B_{iv2}/B_{iv1} ratio given in Ba84.

TABLE C-10 (Continued):

Parameter	Estimated Range of Values	Value Used*	Notes**
F_m (day/liter)			
Ni	1.0E-3 to 1.0E-2	1.0E-3	(Ba84, Pe83)
Sr	3.5E-4 to 3.8E-3	1.5E-3	(Ng82c)
Zr	3.0E-5 to 8.0E-2	3.0E-5	(Ba84, Pe83)
Tc	2.0E-3 to 4.3E-2	1.0E-2	(Ri83)
Sn	1.0E-4 to 1.0E-1	1.0E-3	(Ri83)
I	1.4E-3 to 5.4E-2	1.0E-2	(Ng82c, Ng77)
Cs	1.3E-3 to 3.7E-2	7.0E-3	(Ri83)
Sm	2.0E-5	2.0E-5	(Ba84)
Pb	9.1E-6 to 7.7E-4	2.5E-4	(Ng77)
Ra	9.0E-5 to 7.0E-4	4.5E-4	(Ng77)
Ac	2.0E-5	2.0E-5	(Ba84)
Th	3.0E-6 to 5.0E-6	5.0E-6	(Ng77, Ba84)
Pa	5.0E-6	5.0E-6	(Ba84)
U	7.3E-5 to 6.1E-4	6.0E-4	(Ng77)
Np	1.0E-7 to 1.0E-3	1.0E-5	(Ri83)
Pu	2.7E-9 to 1.0E-7	1.0E-7	(Ng77)
Am	4.0E-7 to 2.0E-5	4.0E-7	(Ba84, Ng77)
Cm	2.0E-5	2.0E-5	(Ba84)

*Values used are default values given by Baes, et. al (Ba84) except for Np which was taken from Rish, et. al (Ri83).

**Notes indicate references used to estimate range of values.

TABLE C-10 (Continued):

Parameter	Estimated Range of Values ***	Value Used*	Notes**
F_f (day/kg)			
Ni	3.4E-4 to 1.1E-2	2.0E-3	(Ng82c)
Sr	2.0E-6 to 8.0E-2	3.0E-4	(Ri83, Ng82c)
Zr	5.5E-3 to 2.0E-2	2.0E-2	(Ba84, Ng82a)
Tc	1.0E-3 to 2.0E-1	8.5E-3	(Ri83, Ng82a)
Sn	1.0E-4 to 1.0E-1	8.0E-2	(Ri83)
I	1.8E-4 to 2.0E-1	7.0E-3	(Ng82a, Ng82c)
Cs	2.9E-3 to 4.5	2.0E-2	(Ri83, Ng82c)
Sm	5.0E-3	5.0E-3	(Ba84)
Pb	1.0E-4 to 2.0E-3	3.0E-4	(Ng82a, Ng82c)
Ra	2.5E-4 to 2.0E-3	5.8E-4	(Ba84, Ng82c)
Ac	1.6E-6 to 2.5E-5	2.5E-5	(Mo79, Ba84)
Th	1.6E-6 to 2.0E-4	6.0E-6	(Mo79, Pe83)
Pa	1.6E-6 to 1.0E-5	1.0E-5	(Mo79, Ba84)
U	1.6E-6 to 1.2	2.0E-4	(Mo79, Ng82a)
Np	1.0E-6 to 5.0E-3	2.0E-4	(Ri83)
Pu	5.0E-9 to 1.6E-4	2.0E-6	(Ng82c, Ng82a)
Am	3.5E-6 to 1.8E-4	3.5E-6	(Ba84, Ng82a)
Cm	3.5E-6	3.5E-6	(Ba84)

*Values used are default values given by Baes, et. al (Ba84) except for Ni, Zr, Ra, Np, and Pu. Values of F_f for these five elements were discussed with C.F. Baes, III (ORNL) and S.A. Schaffer (Envirosphere) and the concensus was:

Ni, Zr, and Pu - use values given by Ng, et. al (Ng82a);
Ra - average the values given by Baes, et. al (Ba84) and Ng, et. al (Ng82a);
Np - values given by Baes, et. al (Ba84) and Ng, et. al (Ng82a) are based on an injection study where gut uptake was not considered. The value should be increased to account for gut uptake.

**Notes indicate references used to estimate range of values.

***For several elements, values of F_f given by Ng, et. al (Ng82a, Ng82c) include data for chicken. F_f for chicken is generally substantially higher than for beef and pork. The "values used" are more representative of beef values. However, for several elements, the upper limit of the "estimated range of values" is for chicken. This is the reason that the "value used" is nearer the lower limit for several elements (see U, for example).

C.10 Determination of Values for the Parameters γ_1, γ_2, SF_{1n} and SF_{2n} Used for the Ocean Release Mode

In this section, we will show the methodology used to determine parameter values which are applied in the algorithms for the ocean release mode. These algorithms are discussed in Chapter 3, sections 3.2 and 3.4.4.

The transfer rate coefficients used in the ocean model are γ_1 and γ_2 (see Figures 3-1 and 3-4). That for movement of water from the lower to the upper ocean compartment is γ_2 and the value of it used for this analysis is 6.25 E-4 yr^{-1}(Ma73). For movement of water from the upper to the lower ocean compartment, the transfer rate coefficient is $\gamma 1$, and the value applied for this analysis is 3.3 E-2 yr^{-1}, which is derived from the expression $\gamma_1 = \gamma_2$ (3925/75) where 3925 m is the assumed depth of the ocean lower compartment and 75 m is the assumed depth of the ocean upper compartment.

The relationship between γ_1 and γ_2 is derived by assuming that interchange of water between the upper and lower layers of the ocean results in no net transfer from one layer to the other, i.e., the mass of each layer remains the same. If we further assume that the density of water in the upper and lower layer is equal close to the boundary between the layers, we can derive a value for γ_1 using the value for γ_2 and using conservation of mass.* The volume of water transport upward per year is equal to $\gamma_2 V_2$ = (6.25 E-4) (3925) (3.61 E14) = 8.9 E14 m^3/yr, where 3.61 E14 m^2 is the area of the oceans (CRC75).

---

*For equal densities close to the boundaries of the two ocean layers, conservation of mass will also result in conservation of volume.

Similarly, the volume of water transported downward per year is equal to $\gamma_1 V_1$ = (3.3 E-2) (75) (3.61 E14) = 8.9 E14 m^3/yr.

Since

$\gamma_1 V_1 = \gamma_2 V_2$, then γ_1 (75) (3.61 E14) =

γ_2 (3925) (3.61 E14) and $\gamma_1 = \gamma_2$ (3925/75).

SF_{1n} and SF_{2n} are the sedimentation coefficients from the upper layer and the lower layer of the ocean, respectively, for nuclide n (see Figures 3-1 and 3-4). The treatment of sedimentation is taken from a report of the working group of the International Nuclear Fuel Cycle Evaluation (INFCE78), which states that the sedimentation coefficients are given by

$$SF_n = \frac{K_{Dn}\, v_s}{h_c} \tag{C-15}$$

where

K_{Dn} = distribution coefficient of the radionuclide on the sediment (dimensionless),

v_s = sedimentation velocity (m/y), given a value of 10^{-5}, and

h_c = average depth of the water column (m) .

Nuclide specific values of SF_{1n} and SF_{2n} can be calculated using the equations

$SF_{1n} = (10^{-5}/75)\, K_{Dn}$ and $SF_{2n} = (10^{-5}/3925)\, K_{Dn}$, or

$$SF_{1n} = 1.33\text{ E-7 } K_{Dn} \tag{C-16}$$

$$SF_{2n} = 2.54\text{ E-9 } K_{Dn}. \tag{C-17}$$

The K_{Dn} values that we used are more conservative than those in INFCE78 to allow for expected competition for adsorption sites by the sea water ions, especially Mg and Ca. In the absence of detailed experimental work on sorption, anionic ions were assigned K_{Dn} values of zero; major sea water ions were given K_{Dn} values of 1; and neptunium was also given a K_{Dn} of 1. The effect of the sea water ions on divalent ions and on cesium was approximated by reducing the desert soil K_{Dn} values given by Arthur D. Little (Li77c) by a factor of 10. Since the monovalent and divalent sea water cations would be expected to have a smaller effect on the adsorption of polyvalent cations, the Arthur D. Little K_{Dn} values were reduced by a factor of 2 for trivalent ions and unchanged for tetravalent ions. The values of K_{Dn} used in this report are listed in Table C-11. Using these values and equations C-16 and C-17, the values for SF_{1n} and SF_{2n} listed in Table 5-6 were computed.

TABLE C-11:
Distribution coefficients for radionuclides on sediment

Element	K_{Dn}(milliliters/g)
C	0
Ni	40
Sr	2
Zr	2000
Tc	0
Sn	0
I	0
Cs	20
Sm (Rare Earth)	300
Pb	2000
Ra	10
Ac	500
Th	15,000
Pa	2000
U	300
Np	1
Pu	2000
Am	1000
Cm	300

APPENDIX D: SAMPLE DERIVATION OF AN ENVIRONMENTAL PATHWAY EQUATION AND CALCULATION OF POPULATION FATAL CANCERS

A derivation of an environmental risk commitment equation and the application of representative data in the equation to compute population health effects for an environmental pathway will serve to illustrate the application of the methodology described in this report. The environmental pathway chosen for demonstration is ingestion of food crops for radionuclide releases to a river (pathway number 3). Terms that are explained in the "Nomenclature" section are not defined again in this Appendix.

An expression for environmental risk commitment must be derived and is the same as is described in section 3.1.4. The starting point is the annual risk commitment to an individual.

The annual risk commitment is given by the concentration of radionuclides in the river water (Q'_{np}/R), the irrigation rate (W), a conversion factor to express the radionuclide intake by an individual per unit deposition to the ground surface (RI_{np}), and the risk conversion factor (FCF_{np}) as

$$RIV'_{np} = \frac{Q'_{np} \; W \; RI_{np} \; FCF_{np}}{R} \quad . \tag{D-1}$$

The annual population risk commitment can be expressed as the individual risk commitment multiplied by the number of persons being fed a particular food crop raised on irrigated land. The number of persons fed can be determined using an estimate of the number of persons who can be fed by raising the food crop on a unit area of land (CP_p), the area of land irrigated (A), and a weighting factor to express the fraction of irrigated land used for a particular crop (f_p) as

$$P_{Fp} = CP_p \; A \; f_p \quad . \qquad \text{(D-2)}$$

Then the annual population risk commitment is

$$FHE'_{np} = RIV'_{np} \; P_{Fp} = \frac{Q'_{np} \; W \; RI_{np} \; FCF_{np} \; CP_p \; A \; f_p}{R} \quad . \qquad \text{(D-3)}$$

By integrating this expression over time, we obtain an equation for environmental risk commitment as

$$FHE_{np} = \frac{Q_{np} \; W \; RI_{np} \; FCF_{np} \; CP_p \; A \; f_p}{R} \quad . \qquad \text{(D-4)}$$

Some rearrangement of terms is desirable in equation D-4 since it is to be used for generic rather than site specific analyses. We can write the relationship

$$W \; A = f_R \; R \qquad \text{(D-5)}$$

which yields

$$\frac{W}{R} = \frac{f_R}{A} \quad . \qquad \text{(D-6)}$$

If equation D-6 is substituted into equation D-4, we obtain

$$FHE_{np} = Q_{np} \; f_R \; f_p \; RI_{np} \; CP_p \; FCF_{np} \quad . \qquad \text{(D-7)}$$

Implicit in equation D-7 is the assumption that all river water used for irrigation contains radionuclides and, consequently, that all land irrigated by river water receives radionuclides released from the waste repository.

Our food pathway calculation will be performed for Tc-99. Parameter values from Chapters 4 and 5 will be used in the sample calculation.

A leaching-rate-limited source-term equation will be used for this illustrative calculation. Figure D-1 shows the predicted travel of radionuclides from the waste repository to the river. An expression for the rate of entry of radionuclide n to the river can be developed after examination of Figure D-1. The inventory of material at time, t_{er}, when leaching begins is

$$Q_{rnp}(t_{er}) = Q_{on}\, e^{-\lambda_{Dn} t_{er}} \tag{D-8}$$

where

$Q_{rnp}(t_{er})$ = inventory of radionuclide n in the repository at the time leaching begins (Ci) .

At any time t_1 ($t_1 \geq t_{er}$) the rate of exit of radionuclide n from the repository is

$$Q'_{rnp}(t_1) = \lambda_{Ln}\, Q_{rnp}(t_{er})\, e^{-(\lambda_{Dn}+\lambda_{Ln})(t_1-t_{er})} \tag{D-9}$$

where

$Q'_{rnp}(t_1)$ = rate of exit of radionuclide n from the repository at time t_1 (where $t_1 \geq t_{er}$) (Ci/yr) .

Now, assuming that radioactive decay is the only mechanism of loss of radionuclide n during travel from the repository to the aquifer (t_{ran})

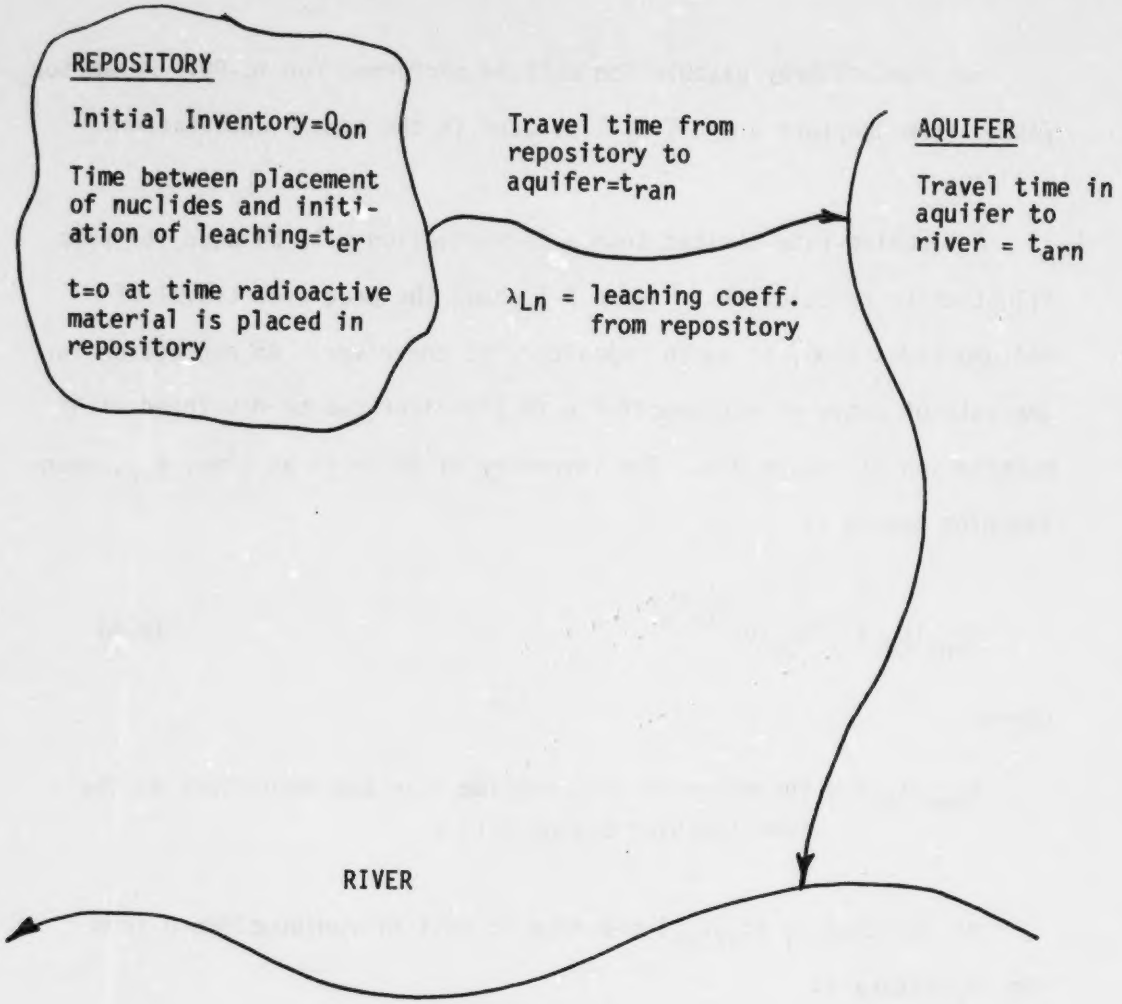

REPOSITORY
Initial Inventory=Q_{on}
Time between placement of nuclides and initiation of leaching=t_{er}
t=o at time radioactive material is placed in repository
Travel time from repository to aquifer=t_{ran}
λ_{Ln} = leaching coeff. from repository
AQUIFER
Travel time in aquifer to river = t_{arn}
RIVER

Fig. D-1. Radionuclide travel from repository to river and travel from the aquifer to the river (t_{arn})* the equation for release rate to the river at any time t is

$$Q'_{np}(t) = Q'_{rnp}(t_1)\, e^{-\lambda_{Dn}(t_{ran}+t_{arn})} \qquad \text{(D-10)}$$

$$\text{for } t > t_{er} + t_{ran} + t_{arn}$$

and

$$Q'_{np}(t) = 0 \qquad \text{for } t \leq t_{er} + t_{ran} + t_{arn} .$$

The relationship between t and t_1 is

$$t = t_1 + t_{ran} + t_{arn} \qquad \text{(D-11)}$$

or

$$t_1 = t - t_{ran} - t_{arn} . \qquad \text{(D-12)}$$

Substituting equations D-8 and D-9 into equation D-10 yields

$$Q'_{np}(t) = \lambda_{Ln}\, Q_{on}\, e^{-\lambda_{Dn}t_{er}}\; e^{-(\lambda_{Dn}+\lambda_{Ln})(t_1-t_{er})}\; e^{-\lambda_{Dn}(t_{ran}+t_{arn})} \qquad \text{(D-13)}$$

or

$$Q'_{np}(t) = \lambda_{Ln}\, Q_{on}\, e^{-\lambda_{Dn}(t_{er}+t_{ran}+t_{arn})}\; e^{-(\lambda_{Dn}+\lambda_{Ln})(t_1-t_{er})} \qquad \text{(D-14)}$$

and substituting equation D-12 into D-14 gives

$$Q'_{np}(t) = \lambda_{Ln}\, Q_{on}\, e^{-\lambda_{Dn}(t_{er}+t_{ran}+t_{arn})}\; e^{-(\lambda_{Dn}+\lambda_{Ln})(t-t_{ran}-t_{arn}-t_{er})} \qquad \text{(D-15)}$$

---

*Other removal mechanisms will be operative during groundwater transport of radionuclides from the respository to a river. However, since our objective is to determine the fatal cancers per Ci released to a river, this simplified transport model is acceptable for these calculations.

or

$$Q'_{np}(t) = \lambda_{Ln}\, Q_{on}\, e^{-\lambda_{Dn} t}\; e^{-\lambda_{Ln}(t-t_{er}-t_{ran}-t_{arn})} \quad . \qquad \text{(D-16)}$$

Now, assuming that only a fraction, f_L, of the contents of the repository are subject to leaching, we can write the final expression for the release rate of radionuclide n to the river as

$$Q'_{np}(t) = \lambda_{Ln}\, f_L\, Q_{on}\, e^{-\lambda_{Dn} t}\; e^{-\lambda_{Ln}(t-t_{er}-t_{ran}-t_{arn})} \qquad \text{for } t > t_{er} + t_{ran} + t_{arn}$$

and (D-17)

$$Q'_{np}(t) = 0 \qquad \text{for } t \leq t_{er} + t_{ran} + t_{arn} \; .$$

To obtain the total (integrated) amount of radionuclide n that has entered the river up to time t, we integrate equation D-17 and get

$$Q_{np}(t) = \int_{o}^{t} Q'_{np}(t'')dt'' \; . \qquad \text{(D-18)}$$

Using equation D-17 and the relationship

$$t_{Rn} = t_{er} + t_{ran} + t_{arn} \qquad \text{(D-19)}$$

we obtain

$$Q_{np}(t) = \frac{Q_{on}\, f_L\, \lambda_{Ln}}{(\lambda_{Dn}+\lambda_{Ln})} \left[e^{-\lambda_{Dn} t_{Rn}} - e^{\lambda_{Ln} t_{Rn}}\, e^{-(\lambda_{Dn}+\lambda_{Ln})t} \right] \qquad \text{for } t > t_{Rn} \; .$$

and (D-20)

$$Q_{np}(t) = 0 \qquad \text{for } t \leq t_{Rn} \; .$$

To determine the total release of Tc-99 we will use the following data:

Q_{on} = 1000 curies of Tc-99

f_L = 0.01

λ_{Ln} = 1.00 E-4 yr^{-1}

λ_{Dn} = 3.27 E-6 yr^{-1}

t_{er} = 100 yrs

t_{ran} = 1 yr

t_{arn} = 760 R_n yrs, where R_n = 1 (Li77c), therefore:

t_{arn} = 760 (1) = 760 yrs.

Now, from equation C-23

$$t_{Rn} = t_{er} + t_{ran} + t_{arn} = 100 + 1 + 760 = 861 \text{ yrs.}$$

Let the time, t, for computation of the health effects commitment be t = 10,000 yrs. Then, using equation D-20 we have

$$Q_{np}(t) = \frac{\lambda_{Ln}\, f_L\, Q_{on}}{\lambda_{Dn} + \lambda_{Ln}} \left[e^{-\lambda_{Dn} t_{Rn}} - e^{\lambda_{Ln} t_{Rn}}\, e^{-(\lambda_{Dn} + \lambda_{Ln})\, t} \right]$$

and

$$Q(10000) = \frac{(1.00E-4)(0.01)(1000)}{(3.27E-6)+(1.00E-4)} \left[e^{-(3.27E-6)(861)} - e^{[(1.00E-4)(861)-(3.27E-6+1.00E-4)(10,000)]} \right]$$

$$Q(10,000) = (9.683)\ (0.997 - 0.388)$$

$Q(10,000)$ = 5.90 curies of Tc-99 released over 10,000 yrs.

To compute a value for RI_{np}, we have the following equation (from Appendix C, Equation C-4).

$$RI_{np} = U^{P} \left(\frac{DDI \cdot f_{cp} \left[1 - e^{\lambda_{En}\, t_{ep}}\right]}{Y_{vp}\, \lambda_{En}} + \frac{B_{iv2} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})\, t_{b}}\right]}{P\, (\lambda_{Dn} + \lambda_{Sn})} \right) e^{-\lambda_{Dn}\, t_{hp}}$$

$$+ U^{L} \left(\frac{DDI \cdot f_{cL} \left[1 - e^{\lambda_{En}\, t_{eL}}\right]}{Y_{vL}\, \lambda_{En}} + \frac{B_{iv1} \left[1 - e^{-(\lambda_{Dn} + \lambda_{Sn})\, t_{b}}\right]}{P\, (\lambda_{Dn} + \lambda_{Sn})} \right) e^{-\lambda_{Dn}\, t_{hL}}$$

The parameter values used in this equation are given in Appendix C (with the references). They are listed again here for completeness.

U^{P} = 176 kg wet/yr　　$DDI = 1.0$

$f_{cp} = 0.052$

$\lambda_{En} = \lambda_{Dn} + \lambda_{w} = 3.26\text{ E-6 yr}^{-1} + 18.41\text{ yr}^{-1} = 18.41\text{ yr}^{-1}$, where

λ_{w} = time constant for loss from vegatation due to weathering, yr^{-1}.

$t_{ep} = t_{eL}$ = 0.274 yrs　　Y_{vp} = 1.6 kg wet/m^{2}

$B_{iv1} = 9.5 \frac{\text{Ci/kg dry crop}}{\text{Ci/kg dry soil}}$　　$B_{iv2} = 0.64 \frac{\text{Ci/kg wet crop}}{\text{Ci/kg dry soil}}$

$\lambda_{Dn} = 3.26\text{ E-6 y}^{-1}$　　$\lambda_{Sn} = 0.49\text{ yr}^{-1}$

t_{b} = 1 E15 yrs*　　P = 215 kg dry soil/m^{2}

$t_{hp} = t_{hL}$ = 3.83 E-2 yr.

U^{L} = 1.2 kg dry/yr　　$f_{cL} = 0.15$

Y_{vL} = 0.12 kg dry/m^{2}

---

*The large value of t_b is used to assure that an equilibrium value for RI_{np} is computed.

Using these values, the value of RI_{np} can be computed as

$$RI_{np} = 176\left(\frac{(1)(.052)\left[1 - e^{-18.41(.274)}\right]}{(1.6)(18.41)} + \frac{(.64)\,[1]}{215(3.26E\text{-}6 + .49)}\right)e^{-(3.26E\text{-}6)(.0383)}$$

$$+ 1.2\left(\frac{(1)(.15)\left[1 - e^{-18.41(.274)}\right]}{(.12)(18.41)} + \frac{9.5[1]}{215(3.26E\text{-}6 + .49)}\right)e^{-(3.26E\text{-}6)(.0383)}$$

$$RI_{np} = 176\,[1.754\ E\text{-}3 + 6.075\ E\text{-}3]\ 1.0 + 1.2[6.746\ E\text{-}2 + 9.018\ E\text{-}2]1.0$$

$$RI_{np} = 1.57\ \frac{\text{Ci intake}}{\text{Ci/m}^2\ \text{deposited}}\ .$$

The remaining parameter values for use in equation D-7 to compute environmental dose commitment are

$f_R = 0.1$* $f_p = 0.5$ $CP_p = 4.8\ E\text{-}3\ \text{man/m}^{2*}$.

$FCF_{np} = 5.4E\text{-}1$ fatal cancers/Ci intake (Appendix B)

The expression for computation of environmental dose commitment is equation D-7, i.e.

$$FHE_{np} = Q_{np}\ f_R\ f_p\ RI_{np}\ CP_p\ FCF_{np}\ . \qquad \text{(D-7)}$$

Substituting the parameter values discussed above into equation D-7 yields

$$FHE_{np} = (5.90)(0.1)(0.5)(1.57)(0.0048)\ (0.54) = 1.19E\text{-}3 \text{ fatal cancers}$$

Thus, for 5.90 Ci of Tc-99 released to the river, the number of fatal cancers in 10,000 yrs to the affected population from consumption of food crops irrigated by contaminated river water is estimated to be 0.00119.

---

*See more detailed discussion in Appendix C.

The fatal cancers per curie released to the accessible environment for this sample calculation are 0.0012/5.90 = 2.02 E-4. The fatal cancers per curie release to the accessible environment for the other nuclides and pathways discussed in this report are determined in a similar manner and are presented in Chapter 6.

APPENDIX E: FORTRAN SOURCE LISTING OF PROGRAM WESPDOSE2: WESP POPULATION ENVIRONMENTAL RISK COMMITMENT CODE

```
C
C  J. M. SMITH
C  EPA/EERF
C  P. O. BOX 3009
C  MONTGOMERY,AL 36193
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/LIST'
C
C  BYTE DIMENSION FOR FILE
      BYTE FILE(30)
C
C  DIMENSION STATEMENT FOR DUMMY VARIABLES FOR PRINTING ALL PATHWAY
C  HEALTH EFFECTS INFORMATION
      DIMENSION PRINTF(MAXP),PRINTG(MAXP)
C
C  DATA WHICH RARELY OR NEVER CHANGES IS CONTAINED IN BLOCK DATA
C  SUBPROGRAM.  READ IN OTHER DATA.
C
C  PROMPT AND TERMINAL READ
C
2     WRITE(5,9[illegible]
      READ(5,90[illegible]PRINT,(XLABEL(J),J=1,30),LTH,FILE
C
C  ASSIGN 30TH [illegible]TE IN FILE TO BE ZERO
C
      FILE(LTH+1)=0
C
C  OPEN FILE AND READ MISC. INPUT DATA AND CLOSE FILE
C
      OPEN(UNIT=1,NAME=FILE,TYPE='OLD',SHARED,READONLY,ERR=2)
C
      READ(1,906)NN,NCARB,FRASUR,(WATER(P),POP(P),
     1USEFR(P),GENVAR(P),PD(P),SOF(P),P=1,MAXP)
      CLOSE(UNIT=1)
C
C  OPEN ALL OUTPUT FILES
C
      OPEN(UNIT=1,NAME='DRO:POPDOSE.SUM',TYPE='NEW')
      OPEN(UNIT=2,NAME='DRO:POPHEF.SUM',TYPE='NEW')
      OPEN(UNIT=3,NAME='DRO:POPHEG.SUM',TYPE='NEW')
      OPEN(UNIT=4,NAME='DRO:DILHEF.SUM',TYPE='NEW')
      OPEN(UNIT=6,NAME='DRO:DILHEG.SUM',TYPE='NEW')
      OPEN(UNIT=7,NAME='DRO:INOUT1.DAT',TYPE='NEW')
      OPEN(UNIT=8,NAME='DRO:INOUT2.DAT',TYPE='NEW')
      OPEN(UNIT=11,NAME='DRO:EGANHEF.SUB',TYPE='NEW')
      OPEN(UNIT=12,NAME='DRO:EGANHEG.SUB',TYPE='NEW')
      IF(IPRINT.EQ.2)OPEN(UNIT=9,NAME='DRO:POPHE.SUB',TYPE='NEW')
C  WRITE OUT APPROPRIATE INPUT DATA AND HEADINGS.
C
```

```
      WRITE(7,909)IPRINT,FILE,IW,IB,NN,MAXO,MAXP,MAXSUM,NWLBOD,
     1NCARB,OVARIE,TESTES,FRASUR,CARCAN,CARGEN,
     2(P,WATER(P),POP(P),USEFR(P),GENVAR(P),PD(P),
     3INTAKE(P),F(P),CP(P),FRCARB(P),SOF(P),IP1(P),IP2(P),IP3(P),P,
     4P=1,MAXP)
C
      WRITE(7,912)((HEAD1(IDUM,O),IDUM=1,2),O=1,MAXO)
      WRITE(8,915)(JDUM,JDUM=1,MAXP2),(JDUM,JDUM=1,MAXP3)
      WRITE(1,918)((HEAD1(IDUM,O),IDUM=1,2),O=1,MAXO)
      WRITE(2,921)
      WRITE(2,927)((HEAD2(IDUM,PDUM),IDUM=1,2),PDUM=1,MAXSUM)
      WRITE(3,924)
      WRITE(3,927)((HEAD2(IDUM,PDUM),IDUM=1,2),PDUM=1,MAXSUM)
      WRITE(4,928)(XLABEL(J),J=1,19)
      WRITE(6,928)(XLABEL(J),J=1,19)
      WRITE(11,970)
      WRITE(11,928)(XLABEL(J),J=1,19)
      WRITE(12,975)
      WRITE(12,928)(XLABEL(J),J=1,19)
      IF(IPRINT.EQ.2)WRITE(9,930)
C
      OPEN(UNIT=10,NAME='DRO:WESP2NUC.DAT',TYPE='OLD',SHARED,READONLY,
     1ERR=2)
C  INITIATE LOOP TO CYCLE THROUGH NUCLIDES
      DO 100 N=1,NN
      PDUM=1
      READ(10,940)(HEAD3(IDUM),IDUM=1,2),DIALNU,(CF(J),J=1,MAXP2),
     1(RI(J),J=1,MAXP3),VG,VW,WTRTRE,GNDCOR,
     2((D(J,O),O=1,MAXO),CHEFAC(J),GHEFAC(J),J=1,MAXP1)
C
      WRITE(7,942)(HEAD3(IDUM),IDUM=1,2),((HEAD4(IDUM,J),IDUM=1,2),
     1(D(J,O),O=1,MAXO),CHEFAC(J),GHEFAC(J),J=1,MAXP1)
C
      WRITE(8,944)(HEAD3(IDUM),IDUM=1,2),DIALNU,N,(CF(J),J=1,MAXP2),
     1(RI(J),J=1,MAXP3),VG,VW,WTRTRE,GNDCOR
C
C  INITIATE LOOP TO CYCLE THROUGH PATHWAYS
C
      DO 70 P=1,MAXP
C
C  INITIATE LOOP TO CYCLE THROUGH ORGANS
C
      DO 50 O=1,MAXO
C
      DOS=DOSN(N,P,O)
      IF(O.EQ.OVARIE.OR.O.EQ.TESTES)DOS=DOS/2.
C
```

```
      DOSSUM(O)=DOSSUM(O)+DOS
50    CONTINUE
C
C  CALCULATE FATAL CANCER INFORMATION
C
      HEFN=HEF(N,P)
      HEFSUM(PDUM)=HEFSUM(PDUM)+HEFN
C
C  CALCULATE SERIOUS GENETIC EFFECTS INFORMATION
C
      HEGN=HEG(N,P)
      HEGSUM(PDUM)=HEGSUM(PDUM)+HEGN
C
      PRINTF(P)=HEFN
      PRINTG(P)=HEGN
C
      IF(IPRINT.EQ.2)WRITE(9,950)(HEAD3(IDUM),IDUM=1,2),P,
     1HEFN,HEGN
C
      IF(P.NE.5.AND.P.NE.8.AND.P.NE.10.AND.P.NE.12.AND.P.NE.15.AND.P
     1.NE.16.AND.P.NE.21.AND.P.NE.22.AND.P.NE.24.AND.P.NE.27.AND.P.NE.
     228.AND.P.NE.30)GO TO 63
C
      WRITE(1,953)(HEAD3(IDUM),IDUM=1,2),(HEAD2(IDUM,PDUM),IDUM=1,2),
     1(DOSSUM(O),O=1,MAXO)
      DO 60 O=1,MAXO
60    DOSSUM(O)=0.
      PDUM=PDUM+1
63    CONTINUE
70    CONTINUE
C
      WRITE(2,956)(HEAD3(IDUM),IDUM=1,2),(HEFSUM(PDUM),PDUM=1,MAXSUM)
      WRITE(4,959)DIALNU,(HEFSUM(PDUM),PDUM=1,MAXSUM)
      WRITE(3,956)(HEAD3(IDUM),IDUM=1,2),(HEGSUM(PDUM),PDUM=1,MAXSUM)
      WRITE(6,959)DIALNU,(HEGSUM(PDUM),PDUM=1,MAXSUM)
      WRITE(11,977)DIALNU,(PRINTF(P),P=1,MAXP)
      WRITE(12,977)DIALNU,(PRINTG(P),P=1,MAXP)
      WRITE(1,963)
      IF(IPRINT.EQ.2)WRITE(9,963)
C
      DO 90 PDUM=1,MAXSUM
      HEFSUM(PDUM)=0.
90    HEGSUM(PDUM)=0.
C
100   CONTINUE
C
```

```
900   FORMAT('   HEY! HEY! HEY!  LET US GET STARTED!!'/1H ,
     1'ON FIRST LINE TYPE IN IPRINT AND THE LABEL',
     2' TO BE PUT ON EGANS FILES.'/1H ,'ON SECOND LINE',
     3' TYPE IN INPUT DATA FILE NAME.')
903   FORMAT(I3,30A4/Q,30A1)
906   FORMAT(I3,2X,I3,2X,E10.3/(6E10.3))
909   FORMAT(36X,'WESP ENVIRONMENTAL DOSE COMMITMENT ESTIMATES'///
     155X,'INPUT DATA'///2X,'CALCULATION OPTION(IPRINT)=',I2,7X,'GENE',
     2'RAL INPUT DATA FILE NAME IS  ',30A1//2X,'IW=',
     31PE10.3,' L/Y     IB=',1PE10.3,' M**3/Y     NN=',I3,4X,'MAXO=',
     4I3,3X,'MAXP=',I3,3X,'MAXSUM=',I3//2X,'NWLBOD=',I3,5X,'NCARB=',
     4I3,5X,'OVARIE=',I3,5X,'TESTES=',I3,5X,'FRASUR=',1PE10.3/
     42X,'CARCAN=',1PE10.3,' FATAL CANCERS/T.B. MAN-REM',5X,
     4'CARGEN=',1PE10.3,' SERIOUS GENETIC EFFECTS/T.B. MAN-REM'///
     552X,'PATHWAY DEPENDENT INPUT DATA'//2X,'P WATER(P)   POP(P)',
     63X,'USEFR(P) GENVAR(P)   PD(P)   INTAKE(P)    F(P)     CP(P)',
     73X,'FRCARB(P)   SOF(P)   IP1  IP2  IP3   P'/
     776X,'PERSONS'/4X,'L/Y OR L',4X,
     8'MAN',17X,'1/M      MAN/M**2     KG/Y',14X,'FED/M**2'/<MAXP>(X,I2,
     91P10E10.3,3(X,I3,X),X,I3/)///)
912   FORMAT(52X,'NUCLIDE DEPENDENT INPUT DATA'//41X,'DOSE EQUIVALENT',
     1' FACTORS AND RISK CONVERSION FACTORS'//4X,'(DOSE UNITS: INHAL.',
     2' AND INGEST.=REM/CI INTAKE, AIR SUBMER.=REM PER CI-Y/M**3 ',
     3'GROUND CONTAM.=REM PER CI-Y/M**2)'/4X,'(RISK UNITS: INHAL. AND',
     4' INGEST.=EFFECTS/CI INTAKE, AIR SUBMER.=EFFECTS PER CI-Y/M**3,',
     5' GROUND CONTAM.=EFFECTS PER CI-Y/M**2)'//124X,'SERIOUS'/
     6115X,'FATAL   GENETIC'/2X,'NUCLIDE   PATHWAY *****',38X,'ORGAN',
     739X,'*****    CANCER  EFFECTS'/20X,A5,A4,2A5,5(A5,A4),2A5,
     82(A5,A4),5X,'RISK     RISK'/116X,'FACTOR   FACTOR')
915   FORMAT(//27X,'**********CF(N,IP2)**********  *********RI(N,IP3)',
     1'**********'/34X,'CI/KG PER CI/L        CI INTAKE PER CI/M**2',
     2' DEPOSITED   VG(N)    VW(N)   WTRTRE(N) GNDCOR(N)'/
     329X,'IP2=',I2,4X,'IP2=',I2,4X,
     3'IP2=',I2,4X,'IP3=',I2,4X,'IP3=',I2,4X,'IP3=',I2,6X,'M/Y',6X,
     4'M/Y'/2X,'NUCLIDE  DIALNU(N)  N'//)
918   FORMAT(/26X,'SMITH FACTORS FOR POPULATION DOSE AS A FUNCTION OF',
     1' NUCLIDE AND ORGAN'//14X,'SUB',46X,'ORGAN'/2X,'NUCLIDE',2X,
     2'PATHWAYS'/12X,'SUMMED',<MAXO-2>(X,2A5),3X,2(X,2A5)/)
921   FORMAT(/33X,'SMITH FACTORS FOR POPULATION FATAL CANCERS',
     1' AS A FUNCTION OF NUCLIDE'/)
924   FORMAT(22X,'SMITH FACTORS FOR POPULATION GENETIC EFFECTS TO ',
     1'FIRST GENERATION AS A FUNCTION OF NUCLIDE'/)
927   FORMAT(/2X,'NUCLIDE  ********************',
     1'*********************************SUBPATHWAYS SUMMED**********',
     2'***************************************'/9X,<MAXSUM>(4X,2A3))
928   FORMAT(1H ,30A4)
```

```
930   FORMAT(17X,'SMITH FACTORS FOR POPULATION HEALTH EFFECTS AS A',
     1' FUNCTION OF NUCLIDE AND SUBPATHWAY'//13X,'SUB',23X,
     2'SERIOUS'/2X,'NUCLIDE  PATHWAY  FATAL',
     3' CANCERS  GENETIC EFFECTS'//)
940   FORMAT(A4,A3,2X,F8.3,3X,<MAXP2+MAXP3+4>(E10.3)/
     1(<MAXO+2>E10.3))
942   FORMAT(/2X,A4,A3,2X,2A4,X,1PE9.2,1PE10.2,1P5E9.2,1PE10.2,
     11P2E9.2,2X,1P2E9.2/(11X,2A4,X,1PE9.2,1PE10.2,1P5E9.2,1PE10.2,
     21P2E9.2,2X,1P2E9.2))
944   FORMAT(2X,A4,A3,3X,F8.3,2X,I2,2X,<MAXP2+MAXP3+4>(1PE10.3))
950   FORMAT(2X,A4,A3,4X,I3,2(6X,1PE10.3))
953   FORMAT(2X,A4,A3,2X,2A3,X,<MAXO-2>(X,1PE10.3),3X,2(X,1PE10.3))
956   FORMAT(2X,A4,A3,2X,<MAXSUM>(1PE10.3))
959   FORMAT(1H ,F8.3,1P6E10.3/1H ,1P6E10.3)
963   FORMAT(/)
970   FORMAT(1X,'FATAL CANCER SMITH FACTORS FOR EACH PATHWAY')
975   FORMAT(1X,'GENETIC EFFECTS--ALL GENERATIONS SMITH FACTORS',
     1' FOR EACH PATHWAY')
977   FORMAT(1H ,F8.3,1P6E10.3/(1H ,1P6E10.3))
      CLOSE(UNIT=1,DISP='SAVE')
      CLOSE(UNIT=2,DISP='SAVE')
      CLOSE(UNIT=3,DISP='SAVE')
      CLOSE(UNIT=4,DISP='SAVE')
      CLOSE(UNIT=6,DISP='SAVE')
      CLOSE(UNIT=7,DISP='SAVE')
      CLOSE(UNIT=8,DISP='SAVE')
      IF (IPRINT.EQ.2)CLOSE(UNIT=9,DISP='SAVE')
      CLOSE(UNIT=10,DISP='SAVE')
      CLOSE(UNIT=11,DISP='SAVE')
      CLOSE(UNIT=12,DISP='SAVE')
      STOP
      END
C
      BLOCK DATA
C  THIS SUBPROGRAM CONTAINS ALL DATA STATEMENTS
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
C  DATA STATEMENTS
      DATA HEAD1/'   BO','NE','RED M','ARROW','   LU','NG','  LIV','ER',
     1'  GI-','LLI','  THY','ROID','  KID','NEY','OTHER','ORGAN',
     2' OVAR','IES','  TES','TES'/
      DATA HEAD2/'  1','-5','  6','-8',' 9-','10',' 11','-12',' 13',
     1'-15','  1','6',' 17','-21','  2','2',' 23','-24',' 25','-27',
     2'  2','8','29','-30'/
      DATA HEAD4/' INH','AL1',' INH','AL2','INGE','ST1','INGE','ST2',
     1'EXT ','AIR','EXT ','GND'/
```

```
       DATA DOSSUM/MAXO*0./
       DATA HEFSUM/MAXSUM*0./
       DATA HEGSUM/MAXSUM*0./
       DATA IP1/4,4,4,4,4,2,6,5,4,4,5,2,4,4,4,6,1,5,3,3,3,6,5,1,3,3,3,
      16,3,3/
       DATA IP2/0,1,0,0,0,0,0,0,2,3,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,
      10,2,3/
       DATA IP3/0,0,1,2,3,0,0,0,0,0,0,0,1,2,3,0,0,0,1,2,3,0,0,0,1,2,3,
      10,0,0/
       DATA FRCARB/0.,0.,1.,0.,0.,0.,0.,0.,1.,0.,0.,0.,1.,0.,0.,0.,
      10.,0.,1.,0.,0.,0.,0.,0.,1.,0.,0.,0.,1.,0./
       DATA NWLBOD,OVARIE,TESTES/8,9,10/
       DATA IW,IB/603.,8401./
       DATA F/0.,0.,.50,.25,.25,0.,0.,0.,0.,0.,0.,0.,.23,.11,.11,0.,0.,
      10.,.23,.11,.11,0.,0.,0.,.23,.11,.11,0.,0.,0./
       DATA CP/0.,0.,.00479,.00156,.0000785,0.,0.,0.,0.,0.,0.,0.,.00479,
      1.00156,.0000785,0.,0.,0.,.00479,.00156,.0000785,0.,0.,0.,.00479,
      2.00156,.0000785,0.,0.,0./
       DATA INTAKE/0.,1.,0.,0.,0.,0.,0.,0.,6.,1.,0.,0.,0.,0.,0.,0.,0.,
      10.,0.,0.,0.,0.,0.,0.,0.,0.,0.,0.,6.,1./
       DATA CARCAN,CARGEN/1.46E-04,7.60E-05/
C
       END
C
       FUNCTION DOSN(N,P,O)
C
C  THIS FUNCTION SUBPROGRAM SELECTS THE PROPER FUNCTION FOR CALCULATING
C  DOSE MULTIPLIER FOR SUBPATHWAY P AND COMPUTES THE DOSE
C  QUANTITY.
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
       IF(N.EQ.NCARB)GO TO 9
30     GO TO (1,2,3,3,3,4,5,6,2,2,6,4,3,3,3,5,4,6,7,7,7,5,6,4,3,
      13,3,5,8,8)P
C
1      DOSN=DOSNA(P)*D(IP1(P),O)
       GO TO 40
2      DOSN=DOSNB(P)*D(IP1(P),O)
       GO TO 40
3      DOSN=DOSNC(P)*D(IP1(P),O)
       GO TO 40
4      DOSN=DOSND(P)*D(IP1(P),O)
       GO TO 40
5      DOSN=DOSNE(P)*D(IP1(P),O)
       GO TO 40
6      DOSN=DOSNF(P)*D(IP1(P),O)
       GO TO 40
7      DOSN=DOSNG(P)*D(IP1(P),O)
```

```
      GO TO 40
8     DOSN=DOSNH(P)*D(IP1(P),0)
      GO TO 40
9     DOSN=DOSNI(P,0)
40    RETURN
      END
C  *************************
C
      FUNCTION HEF(N,P)
C
C  THIS FUNCTION SUBPROGRAM SELECTS THE PROPER FUNCTION FOR CALCULATING
C  FATAL CANCER HEALTH EFFECTS MULTIPLIER FOR SUBPATHWAY P AND
C  COMPUTES THE FATAL CANCERS QUANTITY FOR EGAN'S CODE.
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
      IF(N.EQ.NCARB)GO TO 9
30    GO TO (1,2,3,3,3,4,5,6,2,2,6,4,3,3,3,5,4,6,7,7,7,5,6,4,3,
     13,3,5,8,8)P
C
1     HEF=DOSNA(P)*CHEFAC(IP1(P))
      GO TO 40
2     HEF=DOSNB(P)*CHEFAC(IP1(P))
      GO TO 40
3     HEF=DOSNC(P)*CHEFAC(IP1(P))
      GO TO 40
4     HEF=DOSND(P)*CHEFAC(IP1(P))
      GO TO 40
5     HEF=DOSNE(P)*CHEFAC(IP1(P))
      GO TO 40
6     HEF=DOSNF(P)*CHEFAC(IP1(P))
      GO TO 40
7     HEF=DOSNG(P)*CHEFAC(IP1(P))
      GO TO 40
8     HEF=DOSNH(P)*CHEFAC(IP1(P))
      GO TO 40
C  EQUATION 9 HANDLES FATAL CANCER RISK CALCULATIONS FOR C-14 FOR
C  PATHWAYS 1 THROUGH 30.
C
9     CONTINUE
      IF(P.NE.3.AND.P.NE.9.AND.P.NE.13.AND.P.NE.19.
     1AND.P.NE.25.AND.P.NE.29)GO TO 35
      HEF=CARCAN
      GO TO 40
35    HEF=0.
C
40    RETURN .
      END
C
```

```
      FUNCTION HEG(N,P)
C
C  THIS FUNCTION SUBPROGRAM SELECTS THE PROPER FUNCTION FOR CALCULATING
C  SERIOUS GENETIC EFFECTS TO ALL GENERATIONS MULTIPLIER FOR
C  SUBPATHWAY P AND COMPUTES THE SERIOUS GENETIC EFFECTS QUANTITY
C  FOR EGAN'S CODE.
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
      IF(N.EQ.NCARB)GO TO 9
30    GO TO (1,2,3,3,3,4,5,6,2,2,6,4,3,3,3,5,4,6,7,7,7,5,6,4,3,
     13,3,5,8,8)P
C
1     HEG=DOSNA(P)*GHEFAC(IP1(P))
      GO TO 40
2     HEG=DOSNB(P)*GHEFAC(IP1(P))
      GO TO 40
3     HEG=DOSNC(P)*GHEFAC(IP1(P))
      GO TO 40
4     HEG=DOSND(P)*GHEFAC(IP1(P))
      GO TO 40
5     HEG=DOSNE(P)*GHEFAC(IP1(P))
      GO TO 40
6     HEG=DOSNF(P)*GHEFAC(IP1(P))
      GO TO 40
7     HEG=DOSNG(P)*GHEFAC(IP1(P))
      GO TO 40
8     HEG=DOSNH(P)*GHEFAC(IP1(P))
      GO TO 40
C  EQUATION 9 HANDLES SERIOUS GENETIC RISK CALCULATIONS FOR C-14 FOR
C  P=1 THROUGH 30.
C
9     CONTINUE
      IF(P.NE.3.AND.P.NE.9.AND.P.NE.13.AND.P.NE.19.
     1AND.P.NE.25.AND.P.NE.29)GO TO 35
      HEG=CARGEN
      GO TO 40
35    HEG=0.
C
40    RETURN
      END
C  *************************
C
      FUNCTION DOSNA(P)
C  DRINKING WATER SUBPATHWAY.  P=1
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
```

```
      DOSNA=POP(P)*IW*WTRTRE*FRASUR/WATER(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNB(P)
C  FISH AND SHELLFISH INGESTION.   P=2,9,10
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
      DOSNB=CF(IP2(P))*POP(P)*INTAKE(P)/WATER(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNC(P)
C  ABOVE SURFACE CROPS,MILK,BEEF INGESTION.  P=3,4,5,13,14,15,25,26,27
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
      DOSNC=USEFR(P)*F(P)*RI(IP3(P))*CP(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSND(P)
C  INHALATION OF RESUSPENDED MATERIAL.  P=6,12,17,24
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
      DOSND=GENVAR(P)*PD(P)*IB*USEFR(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNE(P)
C  EXTERNAL DOSE--GROUND CONTAMINATION.  P=7,16,22,28
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
```

```
      DOSNE=USEFR(P)*PD(P)*GNDCOR*SOF(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNF(P)
C  EXTERNAL DOSE--AIR SUBMERSION.  P=8,11,18,23
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
      DOSNF=GENVAR(P)*PD(P)*USEFR(P)*SOF(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNG(P)
C  ABOVE SURFACE CROPS,MILK,BEEF INGESTION.  P=19,20,21
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
      DOSNG=VG*RI(IP3(P))*F(P)*CP(P)*GENVAR(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNH(P)
C  OCEAN FISH AND SHELLFISH INGESTION.  P=29,30
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
C
      DOSNH=CF(IP2(P))*INTAKE(P)*POP(P)*VW*
     1GENVAR(P)/WATER(P)
C
      RETURN
      END
C  ********************
C
      FUNCTION DOSNI(P,O)
C  HANDLES ALL DOSE CALCULATIONS FOR C-14.
C
C  INCLUDE COMMON FILE
 INCLUDE 'DRO:WESPCOM2.FTN/NOLIST'
```

```
      IF(D(IP1(P),NWLBOD).EQ.0..AND.D(IP1(P),O).NE.0.)GO TO 20
      IF(D(IP1(P),NWLBOD).EQ.0..AND.D(IP1(P),O).EQ.0.)GO TO 40
      DOSNI=FRCARB(P)*D(IP1(P),O)/D(IP1(P),NWLBOD)
      GO TO 60
20    WRITE(7,965)
40    DOSNI=0.
965   FORMAT(/2X,'YOU IDIOT--YOU HAVE SET WHOLE BODY DOSE',
     1' FACTOR FOR CARBON TO ZERO WITHOUT SETTING'/2X,'OTHER ',
     2' DOSE FACTORS TO ZERO'/)
C
60    RETURN
      END
C  ********************
C  INFORMATION COMMON TO MAIN CODE AND ALL SUBPROGRAMS
C
C  SET VALUES FOR PARAMETERS
C
      PARAMETER MAXP=30,MAXN=19,MAXO=10,MAXSUM=12,MAXP1=6,MAXP2=3,
     1MAXP3=3
C
C  DEFINE REAL AND INTEGER VARIABLES AND THEIR LENGTHS
C
      INTEGER*2 P,O,PDUM,OVARIE,TESTES
      REAL*4 IW,INTAKE,IB
      REAL*8 HEAD1
C
C  COMMON STATEMENTS
C
      COMMON/BLK1/HEAD1(2,MAXO),HEAD2(2,MAXSUM),HEAD3(2),HEAD4(2,MAXP1),
     1DIALNU,NN,NWLBOD,NCARB,IPRINT,OVARIE,TESTES,XLABEL(30)
C
      COMMON/BLK2/D(MAXP1,MAXO),RI(MAXP3),CF(MAXP2),WATER(MAXP),
     1POP(MAXP),USEFR(MAXP),GENVAR(MAXP),PD(MAXP),INTAKE(MAXP),
     2F(MAXP),CP(MAXP),FRCARB(MAXP),DOSSUM(MAXO),HEFSUM(MAXSUM),
     3HEGSUM(MAXSUM),IP1(MAXP),IP2(MAXP),IP3(MAXP),
     4VG,VW,SOF(MAXP),IW,IB,CARCAN,CARGEN,CHEFAC(MAXP1),GHEFAC(MAXP1),
     5WTRTRE,FRASUR,GNDCOR
```

NOTES PERTINENT TO APPENDICES F, G, AND H.

The information contained in Appendices F, G, and H is adapted from Chapters 7 and 8 and Addendums A and B of the EPA report entitled "Radionuclides: Background Information Document for Final Rules", Volume 1 (EPA84b). These appendices contain their own list of references and definition of symbols and do not utilize the "REFERENCES" and "NOMENCLATURE" sections of the main report.

In our high-level waste environmental pathway calculations, we have used the RADRISK data to convert from radionuclide intake for the inhalation and ingestion pathways and from external exposure for the air submersion and ground surface pathways directly to fatal cancer risk, without calculating dose commitments or dose rates. The methodology applied to compute our fatal cancer risk factors is described in Section 4.1 of Chapter 4. Since the RADRISK computer code utilizes computed dose rates in calculating fatal cancer risks, we have included Appendix F, which describes the radiation dosimetry techniques applied by EPA in these RADRISK calculations.

APPENDIX F: RADIATION DOSIMETRY

F.1 Introduction

Radionuclides transported through the environment may eventually reach people. This contact occurs through either external exposure to radioactive air, water, and ground surfaces or internal exposure from inhaling or ingesting radioactive air, water, or food. Individuals in the population may absorb energy emitted by the decaying radionuclides. The quantification of this absorbed energy is termed dosimetry. This appendix describes the dosimetric models for internal and external exposures, the EPA procedure for implementing the dosimetric equations associated with the models, and the uncertainties in dosimetric calculations.

Mathematical models are used to calculate doses to specific human body organs. The models account for the amount of radionuclides entering the body, the movement of radionuclides through the body, and the energy deposited in organs or tissues resulting from irradiation by the radionuclides that reach the tissue. These models provide the basis for the computer codes, RADRISK and DARTAB, which EPA uses to calculate doses and dose rates. (See Appendix H.)

Uncertainties in dosimetric calculations arise from assumptions of uniform distribution of activity in external sources and source organs and assumptions concerning the movement of the radionuclides in the body. The uncertainties associated with dosimetric calculations are difficult to quantify because the data available for determining distribution for the parameters used in the models are usually insufficient. The major source of uncertainty in dosimetry is the real variation in parameter values among individuals in the general population while doses and dose rates are calculated for a "typical" member of the general population. The three sources of dosimetric uncertainty assessed by EPA are: individual variation, age, and measurement errors. The effects of uncertainty are discussed in greater detail in Section F.6 of this appendix and in Chapter 7.

F.2 Definitions

F.2.1 Activity

Radioactive decay is a process whereby the nucleus of an atom emits excess energy. The emission of this energy is referred to as radioactivity. The "activity" of a radioactive material is characterized by the number of atoms that emit energy, or disintegrate, in a given period of time. The unit of activity used in this report is the picocurie (pCi), which equals 2.22 disintegrations per minute. The excess energy is normally emitted as charged particles moving at high velocities and photons. Although there are many types of emitted radiations, or particles, only three are commonly encountered in radioactive material found in the general environment: alpha radiation (nuclei of helium atoms), beta radiation (electrons), and gamma radiation (photons).

The primary mechanism for radiation damage is the transfer of kinetic energy from the moving alpha and beta particles and photons to living tissue. This transfer leads to the rupture of cellular constituents resulting in electrically charged fragments (ionization). Although the amount of energy transferred is small in absolute terms, it is enough to disrupt the molecular structure of living tissue, and, depending on the amount and location of the energy release, leads to the risk of radiation damage.

F.2.2 Exposure and Dose

The term "exposure" denotes physical contact with the radioactive material. The term "dose" refers to the amount of energy absorbed per gram of absorbing tissue as a result of the exposure. An exposure, for example, may be acute, i.e., occur over a short period of time, while the dose, for some internally deposited materials, may extend over a long period of time.

The dose is a measure of the amount of energy deposited by the alpha and beta particles or photons and their secondary radiations in the organ. The only units of dose used in this chapter are the rad--defined as 100 erg (energy units) per gram (mass unit)--and the millirad (mrad), which is one one-thousandth of a rad. The rad represents the amount, on average, of potentially disruptive energy transferred by ionizing radiation to each gram of tissue. Because it is necessary to know the yearly variation in dose for the calculations described in this report, the quantity used will be the average annual dose (or dose rate) in rad or millirad (per year).

F.2.3 External and Internal Exposures

Radiation doses may be caused by either external or internal exposures. External exposures are those caused by radioactive materials located outside the body, such as irradiation of the body by radioactive material lying on the ground or suspended in the air. Internal exposures are caused by radioactive material that has entered the body through the inhalation or consumption of radioactive material. Having once entered the body, the contaminant may be transmitted to other internal organs and tissues.

The external exposures considered in this report are those resulting from irradiation of the body by gamma rays only. Gamma rays (high energy photons) are the most penetrating of those radiations considered and external gammas may normally contribute to the radiation dose affecting all organs in the body. Beta particles (electrons), which are far less penetrating, normally deliver their dose to, or slightly below, the unshielded surface of the skin and are not considered because their impact is small, particularly on clothed individuals. Alpha particles (helium nuclei), which are of major importance internally, will not penetrate unbroken skin and so are also excluded from the external dose calculations. The internal exposures considered in this report originate from all three types of radiation.

F.2.4 Dose Equivalent

Different types of charged particles differ in the rate at which their energy is transferred per unit of length traveled in tissue, a parameter called the linear energy transfer (LET) of the particle. Beta particles generally have a much lower LET than alpha particles. Alpha particles are more damaging biologically, per rad, than gamma rays and beta particles. In radiation protection, this difference is accounted for by multiplying the absorbed dose by a modifying factor, Q, the quality factor, to obtain a dose equivalent. The quality factor is intended to correct for the difference in LET of the various particles. At present, the International Commission on Radiological Protection (ICRP77) recommends the values Q=1 for gamma rays and beta particles and Q=20 for alpha particles. The units for the dose equivalent, corresponding to the rad and millirad, are rem and millirem. Thus, dose equivalents for gamma rays and beta particles are numerically equal to the dose since the dose equivalent (mrem) = (Q=1) x dose (mrad) while alpha dose equivalents are twenty times as large, dose equivalent (mrem) = (Q=20) x dose (mrad).

F.3 Dosimetric Models

The radiation dose has been defined, in Section F.2.2, as the amount of energy absorbed per unit mass of tissue. Calculation of the dose requires the use of mathematical models such as that shown later in Equation (F-2). In this equation, the amount of activity ingested, I, is multiplied by the fraction, f_1, going to the blood, and the fraction, f'_2, going to a specific tissue. E is the amount of energy absorbed by the tissue for each unit of activity so that the product of all these factors divided by the mass of the tissue is, by definition, the radiation dose per unit activity. The remaining term, $[1-e^{-\lambda t}]/\lambda$, indicates how the activity deposited in the tissue changes with time. All these factors together yield the dose rate. A more comprehensive description of the equations used is given in Appendix H.

F.3.1 Internal Doses

Any effort at calculating dose and risk must, of necessity, involve the use of models. In its simplest form, a model is a mathematical representation of a physical or biological system. If, for example, the amount of radioactive material in an organ is measured periodically, a graph of the activity in the organ, such as that in Figure F-1, is obtained. In the simplest case, analysis of these data may indicate that the fraction of the initial activity, R, retained in the organ at any time, t, is given by an equation of the form

$$R = e^{-\lambda t} \tag{F-1}$$

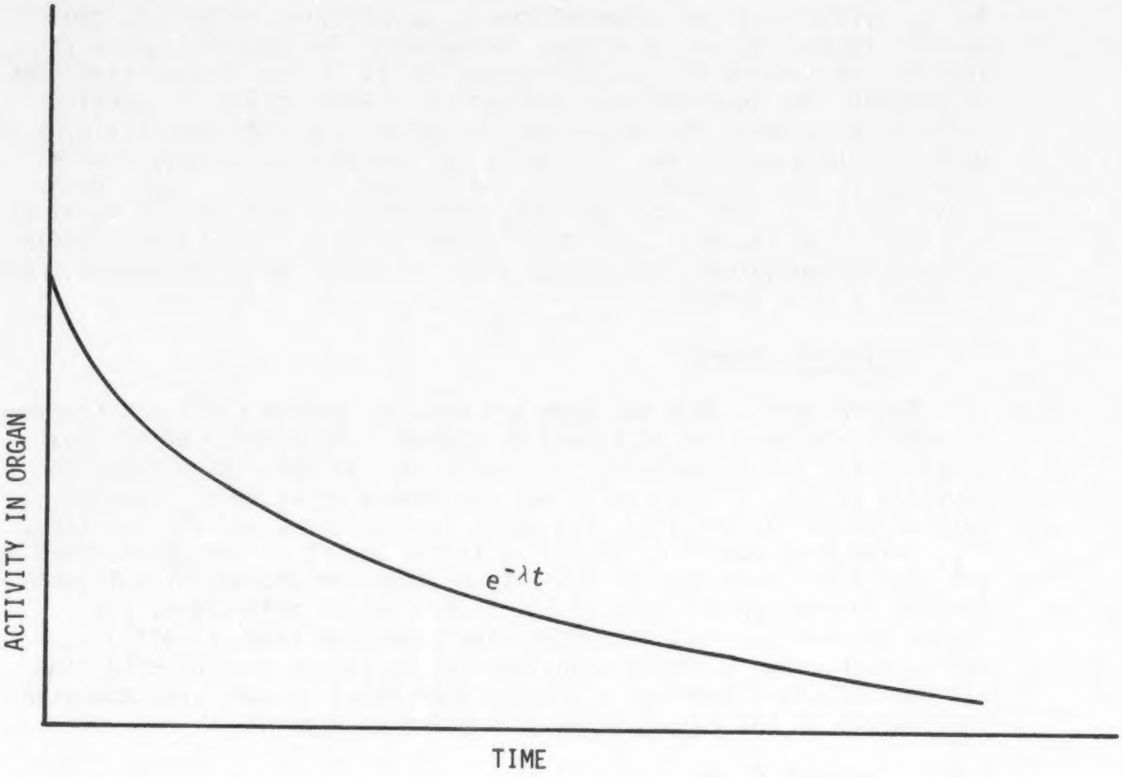


Figure F-1. Typical pattern of decline of activity of a radionuclide in an organ, assuming an initial activity in the organ and no additional uptake of radionuclide by the organ (ORNL81) where λ is the elimination rate constant. (More generally, it may require the sum of two or more exponential functions to properly approximate the decrease of radioactivity in the organ. This may be interpreted physically as indicating the existence of two or more "compartments" in the organ from which the radionuclide leaves at different rates.)

The elimination rate constant, λ, is the sum of two terms, which may be measured experimentally, one proportional to the biological clearance half-life and the other proportional to the radioactive half-life. The effective half-life, $t_{1/2}$, for these processes is the time required for one-half of the material originally present to be removed by biological clearance and radioactive decay.

If radionuclides are generally found to follow this behavior, then this equation may be used as a general model for the activity in an organ following deposition of any initial activity. In general, the models used by EPA are those recommended by the International Commission on Radiological Protection (ICRP79) and are documented in detail in the cited reference. A brief description of each model is given below as an aid to understanding the material presented in the balance of this chapter.

As mentioned earlier, all radiations--gamma, beta, and alpha--are considered in assessing the doses resulting from internal exposure, that is, exposure resulting from the inhalation or ingestion of contaminated material. Portions of the material inhaled or ingested may not leave the body for a considerable period of time (up to decades); therefore, dose rates are calculated over a corresponding time interval.

The calculation of internal doses requires the use of several models. The most important are the ICRP lung model, depicted in Figure F-2, and the gastrointestinal (GI) tract model shown in Figure F-3. The lung model is comprised of three regions, the nasopharyngial (N-P), the tracheobronchial (T-B), and the pulmonary (P) regions. A certain portion of the radioactive material inhaled is deposited in each of the three lung regions (N-P, T-B, and P) indicated in Figure F-2. The material is then cleared (removed) from the lung to the blood and gastrointestinal tract, as indicated by the arrows, according to the specified clearance parameters for the clearance class of the inhaled material.

Deposition and clearance of inhaled materials in the lung are controlled by the particle size and clearance class of the material. The particle size distribution of the airborne material is specified by giving its Activity Median Aerodynamic Diameter (AMAD) in microns (one micron equals 10^{-6} meters). Where no AMAD is known, a value of 1.0 micron is assumed. Clearance classes are stated in terms of the time required for the material to leave the lung, that is, Class D (days), Class W (weeks), and Class Y (years).

Compartment		Class					
		D		W		Y	
		T	F	T	F	T	F
N-P	a	0.01	0.5	0.01	0.1	0.01	0.01
(D_3 = 0.30)	b	0.01	0.5	0.4	0.9	0.4	0.99
T-B	c	0.01	0.95	0.01	0.5	0.01	0.01
(D_4 = 0.08)	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	n.a.	n.a.	1.0	0.4	1.0	0.4
(D_5 = 0.25)	g	n.a.	n.a.	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9

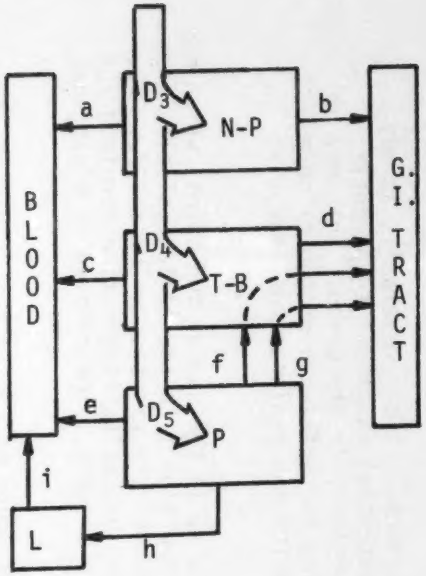


Figure F-2. The ICRP Task Group lung model for particulates

The columns labeled D, W, and Y correspond, respectively, to rapid, intermediate, and slow clearance of the inspired material (in days, weeks, or years). The symbols T and F denote the biological half-time (days) and coefficient, respectively, of a term in the appropriate retention function. The values shown for D_3, D_4, and D_5 correspond to activity median aerodynamic diameter AMAD = 1 μm and represent the fraction of the inspired material depositing in the lung regions.

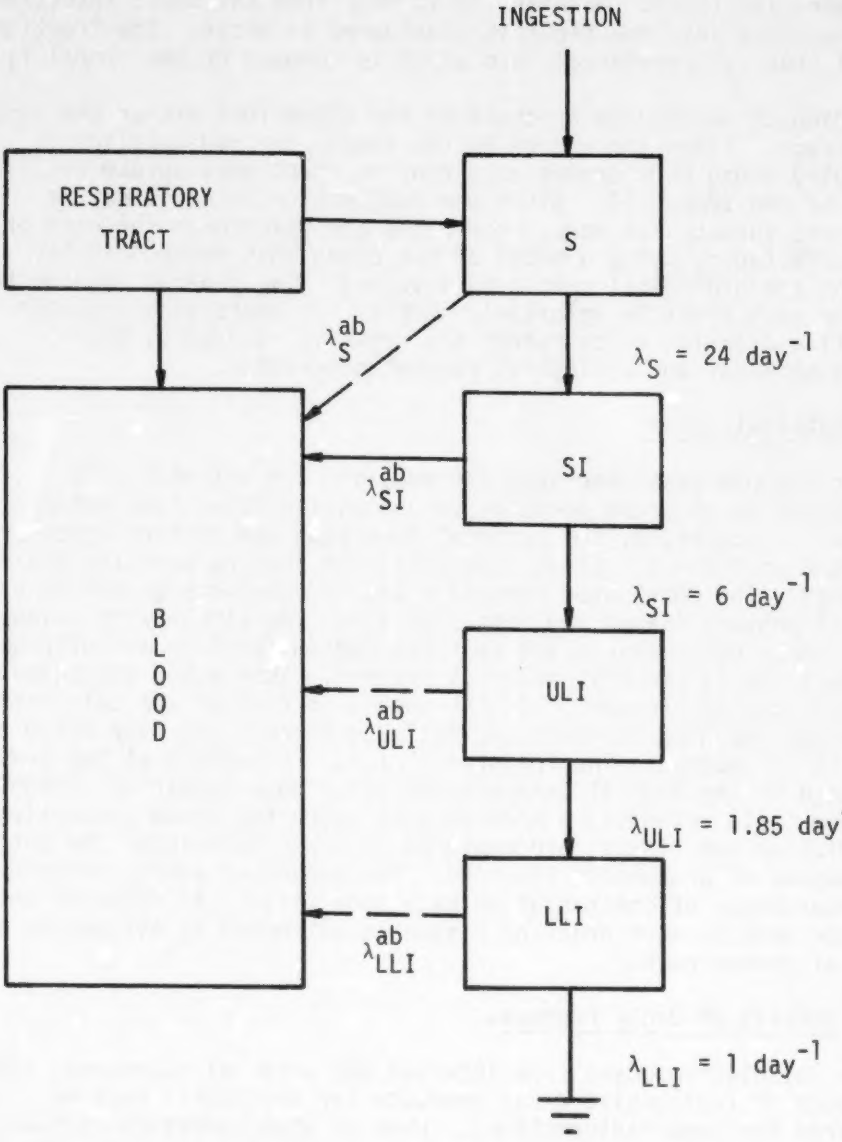

INGESTION
RESPIRATORY TRACT
S
λ_S^{ab}
$\lambda_S = 24\ \mathrm{day}^{-1}$
B L O O D
SI
λ_{SI}^{ab}
$\lambda_{SI} = 6\ \mathrm{day}^{-1}$
ULI
λ_{ULI}^{ab}
$\lambda_{ULI} = 1.85\ \mathrm{day}^{-1}$
LLI
λ_{LLI}^{ab}
$\lambda_{LLI} = 1\ \mathrm{day}^{-1}$

Figure F-3. Schematic representation of radionuclide movement among respiratory tract, gastrointestinal tract, and blood

S = stomach
SI = small intestine
ULI = upper large intestine
LLI = lower large intestine
λ = elimination rate constant

The gastrointestinal tract model consists of four compartments, the stomach (S), small intestine (SI), upper large intestine (ULI), and lower large intestine (LLI). However, it is only from the small intestine (SI) that absorption into the blood is considered to occur. The fraction of material that is transferred into blood is denoted by the symbol f_1.

Radionuclides may be absorbed by the blood from either the lungs or the GI tract. After absorption by the blood, the radionuclide is distributed among body organs according to fractional uptake coefficients, denoted by the symbol f_2'. Since the radioactive material may be transported through the body, dose rates are calculated for each organ or tissue affected by using a model of the organ that mathematically simulates the biological processes involved. The general form of the model for each organ is relatively simple. It postulates that the radioactive material which enters the organ is removed by both radioactive decay and biological removal processes.

F.3.2 External Doses

The example just described for modeling the activity of a radionuclide in an organ pertains to estimating doses from internal exposure. In contrast, the external immersion and surface doses are calculated as follows. First, the number of photons reaching the body is determined. The model used here is a set of equations governing the travel of photons (gamma radiation) in air. The simplifying assumptions used in these calculations are that the medium (air) is an infinite half-space and is the only material present. This makes the calculation relatively straightforward. In the second portion of the calculation, the photons reaching the body are followed through the body using a "Monte Carlo" method. The "phantoms," i.e., the models of the body, are those used by the Medical Internal Radiation Dose Committee (MIRD69). The Monte Carlo method is a procedure in which the known properties of the radiation and tissues are employed to trace (simulate) the paths of a large number of photons in the body. The amount of energy released at each interaction of the radiation with body tissues is recorded and, thus, the dose to each organ or tissue is estimated by evaluating a large number of photon paths.

F.3.3 Effects of Decay Products

In calculating doses from internal and external exposures, the occurrence of radioactive decay products (or daughters) must be considered for some radionuclides. When an atom undergoes radioactive decay, the new atom created in the process may also be radioactive and may contribute to the radiation dose. Although these decay products may be treated as independent radionuclides in external exposures, the decay products of each parent must be followed through the body in internal exposures. The decay product contributions to the dose rate are included in the dose calculations, based on the metabolic properties of the element and the organ in which they occur.

F.3.4 Dose Rate Estimates

For each external and internal exposure, dose rates to each of the organs listed in Table F-1 are calculated for each radioisotope. These organ dose rates serve as input to the life table calculations described in Appendix G.

TABLE F-1:
Organs for which dose rates are calculated

Red bone marrow
Bone
Lung
Breast
Stomach
Pancreas
Intestine
Thyroid
Liver
Urinary tract
Other[a]

[a] Esophagus, lymphatic system, pharynx, larynx, salivary gland, brain.

F.4 EPA Dose Calculation

F.4.1 Dose Rates

The models described in Section F.2 are used by EPA to calculate radiation dose rates resulting from internal and external exposures to radioactive materials. A more complete description of the methodology, equations, and parameters used is given in Du84, ORNL80, and ORNL81. EPA has adopted two refinements to the ICRP-recommended protocol for these calculations. The first is to track the movement of internally produced radioactive daughters by assuming that their movement is governed by their own metabolic properties rather than those of the parent. Although not enough information is available to allow a rigorously defensible choice, this appears to be more accurate for most organs and radionuclides than the ICRP assumption that daughters behave exactly as the parent. In the second departure from ICRP recommendations, age-dependent values of the parameters governing the uptake of transuranic radionuclides have been taken from two sources, deemed appropriate to the general population, the National Radiological Protection Board (NRPB82) and the EPA transuranic guidance document (EPA77).

The internal dose equations given by ICRP may be used to calculate either radiation doses (rad), i.e., the total dose over a given time period, or radiation dose rates (rad/yr), i.e., the way in which the dose changes with time after intake. The integral of the dose rates is, of course, the total dose. EPA calculates dose rates rather than doses, because EPA considers age when assessing the effects of radiation on the population.

External irradiation does not result in any residual internal material. Therefore, external dose rates to a given organ are constant as long as the external radionuclide is present. That is, the dose rate caused by a given amount of radionuclide present in air or on a ground surface becomes zero when the radionuclide is removed.

The calculation of dose rates, rather than integrated doses, allows the use of age-dependent metabolic parameters more appropriate to the general population to be taken into account. In the vast majority of cases, however, there is not now sufficient information available to make such calculations. The effect of using age-dependent metabolic parameters is discussed in Section F.5.2 for some radionuclides for which sufficient information is available.

F.4.2 Exposure and Usage

The ICRP dosimetric equations used by EPA are linear, i.e., an intake of 10 picocuries will result in dose rates ten times as large as those from an intake of 1 picocurie. In similar fashion, exposure to 10 times as large an air or ground surface concentration will increase the external doses by a factor of 10. EPA uses this linearity to avoid having to calculate radiation dose rates for a range of concentrations. The standard EPA procedure is to use unit intakes of 1 pCi/yr and air and ground surface concentrations of 1 pCi/cm^3 and 1 pCi/cm^2, respectively. The doses for other intakes and concentrations may then be scaled up or down as required.

In most cases, it is necessary to make certain assumptions regarding the exposure conditions in order to perform an assessment. EPA calculates dose rates for lifetime exposure to the unit intakes and concentrations. Appendix G describes the different ways in which these rates can be applied. In addition, the exposure assessment will usually depend on other usage conditions assumed for the exposures.

F.5 Uncertainty Analysis

Uncertainty, in the dose, refers to the manner in which the calculated dose changes when the parameters used in the calculation (intakes, metabolic factors, organ sizes, etc.) are changed. The uncertainty associated with the dosimetric calculations is extremely difficult to quantify because the term "uncertainty analysis" implies a knowledge of parameter distributions that is usually lacking. Internal doses, for example, depend on the parameters used to characterize the physiological and metabolic properties of an individual, while external doses must consider parameters such as organ size and geometry for a particular individual. The data available for most of these parameters is not sufficient to define the form of the parameter distribution.

The major source of uncertainty in calculating the dose to a distinct individual, however, in most instances, does not result from errors in measuring the parameters but from the real variation in parameter values among individuals in the general population. Thus, a calculated dose is thought to be representative of a "typical" member of the general population and is probably reasonably precise for some large segment of that population.

The basic physiological and metabolic data used by EPA in calculating radiation doses are taken from the ICRP Report of the Task Group on Reference Man (ICRP75) and from the ICRP Limits for Intakes for Radionuclides by Workers (ICRP79). The "Reference Man" report is the most comprehensive compilation of data available on the intake, metabolism, internal distribution, and retention of radioisotopes in the human body. Its major purpose, however, is to "define Reference Man, in the first instance, as a typical occupational individual," although differences with respect to age and sex are indicated in some instances.

The limitations inherent in defining Reference Man, and in estimating uncertainties due to variations in individuals in the general population, are recognized by the Task Group (ICRP75):

> "The Task Group agreed that it was not feasible to define Reference Man as an 'average' or a 'median' individual of a specified population group and that it was not necessary that he be defined in any such precise statistical sense. The available data certainly do not represent a random sample of any specified population. Whether the sample is truly representative of a particular population group remains largely a matter of judgement which cannot be supported on the basis of statistical tests of the data since the sampling procedure is suspect. Thus the Task Group has not always selected the 'average', or the 'median', of the available measurements in making its selection, nor has it attempted to limit the sample to some national or regional group and then seek an average or median value. However, the fact that Reference Man is not closely related to an existing population is not believed to be of any great importance. If one did have Reference Man defined precisely as having for each attribute the median value of a precisely defined age group in precisely limited locality (e.g., males 18-20 years of age in Paris, France, on June 1, 1964), these median values may be expected to change somewhat with time, and in a few years may no longer be the median values for the specified population. Moreover, the Reference Man so defined would not have this relation to any other population group unless by coincidence. To meet the needs for which Reference Man is defined, this precise statistical relationship to a particular population is not necessary. Only a very few individuals of any population will have characteristics which approximate closely those of Reference Man, however he

> is defined. The importance of the Reference Man concept is that his characteristics are defined rather precisely, and thus if adjustments for individual differences are to be made, there is a known basis for the dose estimation procedure and for the estimation of the adjustment factor needed for a specified type of individual."

With respect to the dosimetric calculations performed by EPA to assess the impact of radioactive pollutants on a general population, three sources of uncertainty should be considered:

(1) that due to the variation in individual parameters among adults in the general population

(2) that due to the variation in individual parameters with age

(3) that due to experimental error in the determination of specific parameters

Each of these sources of uncertainty is discussed in this section. As noted above, the data required to perform a rigorous uncertainty analysis are lacking, and a form of uncertainty analysis called sensitivity analysis is employed. The sensitivity analysis consists of substituting known ranges in the parameters for the recommended value and observing the resulting change in the calculated dose rate.

F.5.1 Dose Uncertainty Resulting from Individual Variation

This section discusses the uncertainty in calculated radiation doses occasioned by differences in physical size and metabolism among individuals in the general population. In order to investigate the effects of individual differences in intake, size, and metabolism, it is necessary to consider the form of the equation used to calculate radiation dose rates. Equation F-2 is a simplified form of the one used by EPA to represent the ingestion of radioactive materials.

$$\dot{D}(t) = c\ I\ f_1\ f_2'\ \frac{E}{m}\ \frac{1}{\lambda}\ [1-e^{-\lambda t}] \qquad \text{(F-2)}$$

where $\dot{D}$ is the dose rate (mrem/yr)

I is the intake rate of radioactivity (pCi/yr)

f_1 is the fraction of I transferred to blood after ingestion

f_2' is the fraction transferred to an organ from the blood

m is the mass of the organ (g)

λ is the elimination constant, which denotes how rapidly the activity is removed from the organ (yr^{-1})

E is the energy absorbed by the organ for each radioactive disintegration (ergs)

c is a proportionality constant.

For simplicity, we will assume that dose rates at large times, t, are to be studied so that the term in the bracket is approximately unity.

Although the actual equations used are considerably more complicated because they must describe the lung model and the GI tract, and also treat all radioactive progeny, the essential features of the uncertainty in dose calculation are reflected in the terms of Equation (F-2). The sensitivity of the dose to each of the terms in the equation may be studied by substituting observed ranges of the quantities for the single value recommended by Reference Man. For some of these quantities, as noted below, no range is cited because of insufficient data.

Intake, I

As an example, postulate that the ingestion mode to be calculated is for fluid intakes. The average fluid intake is about 1900 ml, with an adult range of 1000 to 2400 for "normal" conditions. Under higher environmental temperatures, this range may be increased to 2840 to 3410 ml. Thus, a dose calculated as 1.9, for example, could range from 1.0 to 2.4.

Transfer Fraction, f_1

The value of the transfer fraction to blood depends on the chemical form of the element under study. One of the most common naturally occurring radionuclides is uranium, which is used here as an example. ICRP79 cites values of f_1 ranging from 0.005 to 0.05 for industrial workers, but notes that a higher value of 0.2 is indicated by dietary data from persons not occupationally exposed. EPA has used the 0.2 value for the general population but, based on the ICRP range above, a calculated dose determination could vary by a factor of 10.

Organ Mass, m

The range of organ masses depends primarily on the organ under investigation. For example, reported values for the bloodless lungs range from 461 to 676 grams. Liver weights ranged from 1400 to 2300 grams for adult males and 1200 to 1820 grams for females. Thus, because the organ mass appears in the denominator, calculated lung doses might be expected to vary by a factor of 1.5 and liver doses by a factor of about 2.

Remaining Terms, f_2', λ, E

There are few reported data on the ranges in values to be expected for the remaining variables. They are all quantities which are less directly observable than I, f_1, and m and their influence on the dose calculation can only be estimated. The discussion in Section F.6 is intended to augment the uncertainty analysis by introducing the results of some direct observations on segments of the general population.

F.5.2 Dose Uncertainty Resulting from Age

The dose rates calculated by EPA are normally based on the metabolism and physical characteristics of Reference Man (ICRP75). These properties may obviously be expected to depend on the age of an individual. Most particularly, for infants and children such factors as breathing rates, liquid and solid intakes, organ size and growth rates, and body geometry are known to vary considerably from adult values. The effect of such changes on the radiation dose also depends on the chemistry of the radioactive element under study. For example, rapid bone growth in children is of more importance when a "bone seeker" such as strontium is considered. Although the data available for most age and chemical element combinations are insufficient to allow estimation of the uncertainty in dose, some organ/element combinations, for which more information is available, are discussed below.

Iodine and the Thyroid

Iodine is rapidly and virtually completely absorbed into the bloodstream following inhalation or ingestion. From the blood, iodine enters the extracellular fluid and quickly becomes concentrated in the salivary, gastric, and thyroid glands. It is rapidly secreted from the salivary and gastric glands, but it is retained in the thyroid for relatively long periods.

The intake and metabolism of iodine have been reviewed extensively (ORNL84a) to develop an age-dependent model for iodine. In the model used here, ingested iodine is assumed to be almost completely absorbed by the blood. The remaining parameters are age dependent and are shown in Table F-2. The fluid intake varies from 0.72 liters per day for a newborn to about 2.0 liters per day for an adult.

These age-dependent parameters may then be used in Equation (F-2) to calculate the dose rate resulting from a constant concentration of iodine in water and air. The resulting curves for the dose rate as a function of age are shown in Figures F-4 and F-5. These may be compared to the dose rates obtained using Reference Man parameters at all ages, indicated by the dotted lines in the same figures. Thus, for this particular combination of organ and isotope, the total (70-year) dose is seen to increase by about 30 percent for ingestion and 35 percent for inhalation when dependence on age is considered.

Strontium and Bone

Because of the chemical similarities of strontium and calcium, strontium tends to follow the calcium pathways in the body and deposits to a large extent in the skeleton. In fact, the fraction of ingested strontium eventually reaching the skeleton at a given age depends largely on the skeletal needs for calcium at that age, although the body is able to discriminate somewhat against strontium in favor of calcium after the first few weeks of life.

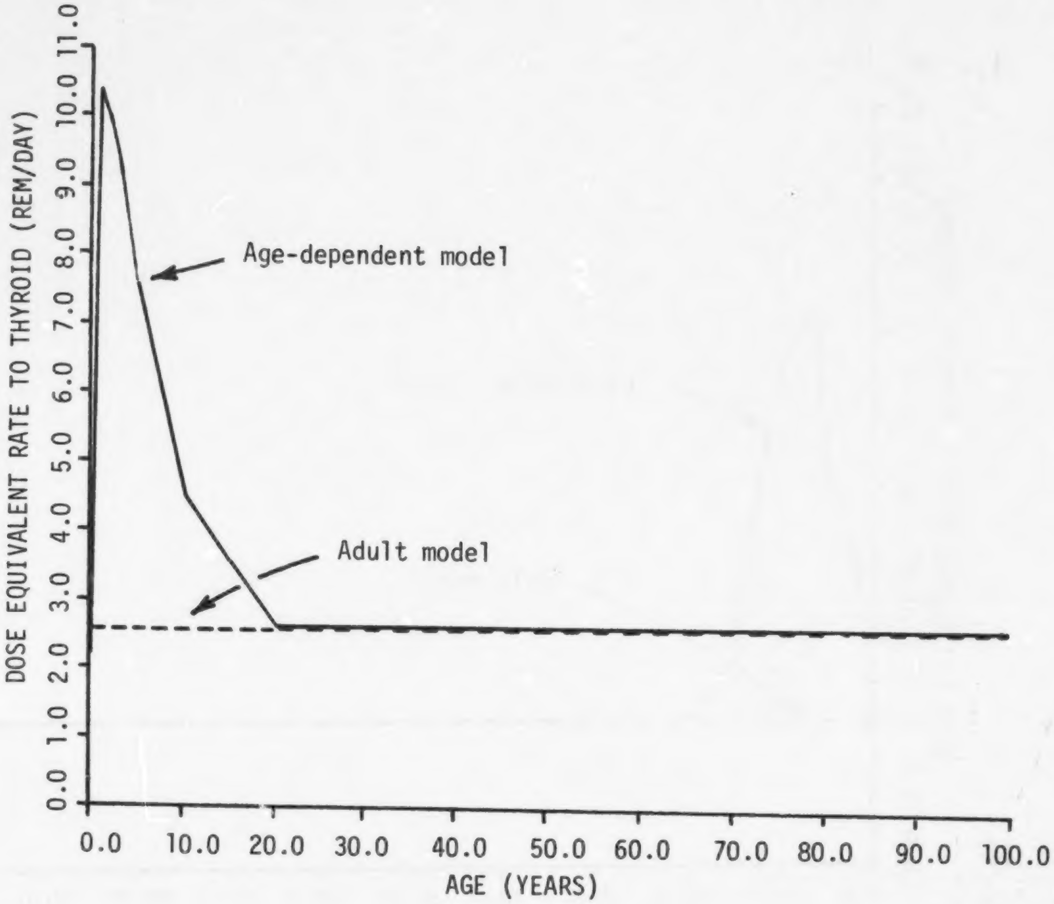


Figure F-4. Dose rate from chronic ingestion of iodine-131 in water at a concentration of 1 μCi/l

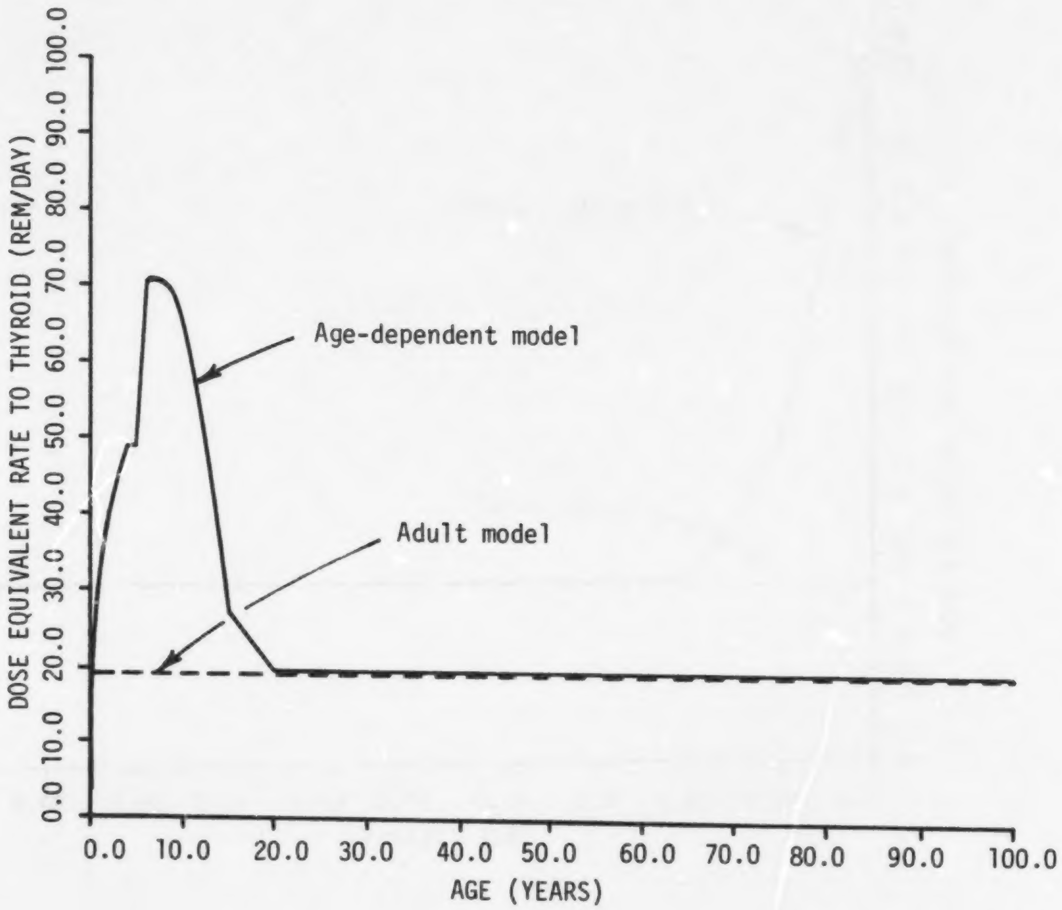


Figure F-5. Dose rate from chronic inhalation of iodine-131 in air at a concentration of 1 μCi/m^3

TABLE F-2:
Age-dependent parameters for iodine metabolism in the thyroid

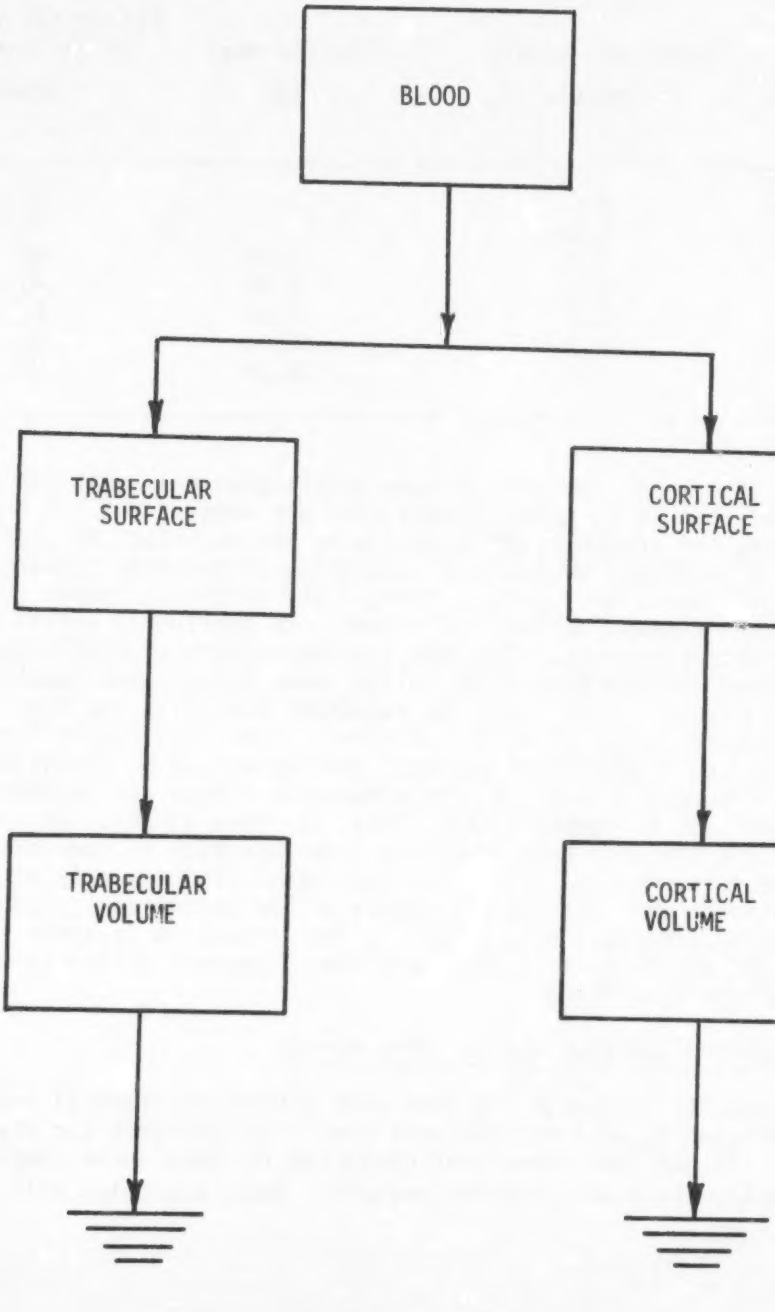
Age (days)	Fractional uptake to thyroid, f_2'	Thyroid mass (g)	Biological half-time in the thyroid (days)
Newborn	0.5	-	15
100	0.40	-	20
365	0.3	1.78	30
1825	0.3	3.45	40
3650	0.3	7.93	50
5475	0.3	12.40	65
7300	0.3	20.00	80

The ICRP model for bone is more complicated than that for the thyroid because it consists of more than one compartment. For purposes of modeling the transport of strontium by the skeleton, it suffices to view the mineralized skeleton as consisting of two main compartments: trabecular (cancellous, porous, spongy) and cortical (compact) bone. Two subcompartments, surface and volume, are considered within each of these main compartments. The four subcompartments of mineralized skeleton and the movement of strontium among these compartments are shown schematically in Figure F-6. The equations governing the age dependence of the parameters are given in (ORNL84a). Dose rate curves for the inhalation and ingestion of constant concentrations of strontium-90 are given in Figures F-7 and F-8 The comparable curves for Reference Man are again indicated by dashed lines. Thus, for this element and organ combination, the dose rate resulting from ingestion is somewhat higher, while the dose rate resulting from inhalation exhibits only minor perturbations, when the age dependence of the parameters is considered. The lifetime (70-year) dose resulting from ingestion is about 7 percent greater and the inhalation dose less than 1 percent different when age dependence is considered.

Plutonium and Lung and Red Bone Marrow

Apparently plutonium and iron bear sufficient chemical resemblance that plutonium is able to penetrate some iron transport and storage systems. It has been shown that plutonium in blood serum complexes with transferrin, the iron-transport protein. Thus, plutonium will partially

Figure F-6. Compartments and pathways in model for strontium in skeleton


BLOOD
TRABECULAR SURFACE
CORTICAL SURFACE
TRABECULAR VOLUME
CORTICAL VOLUME

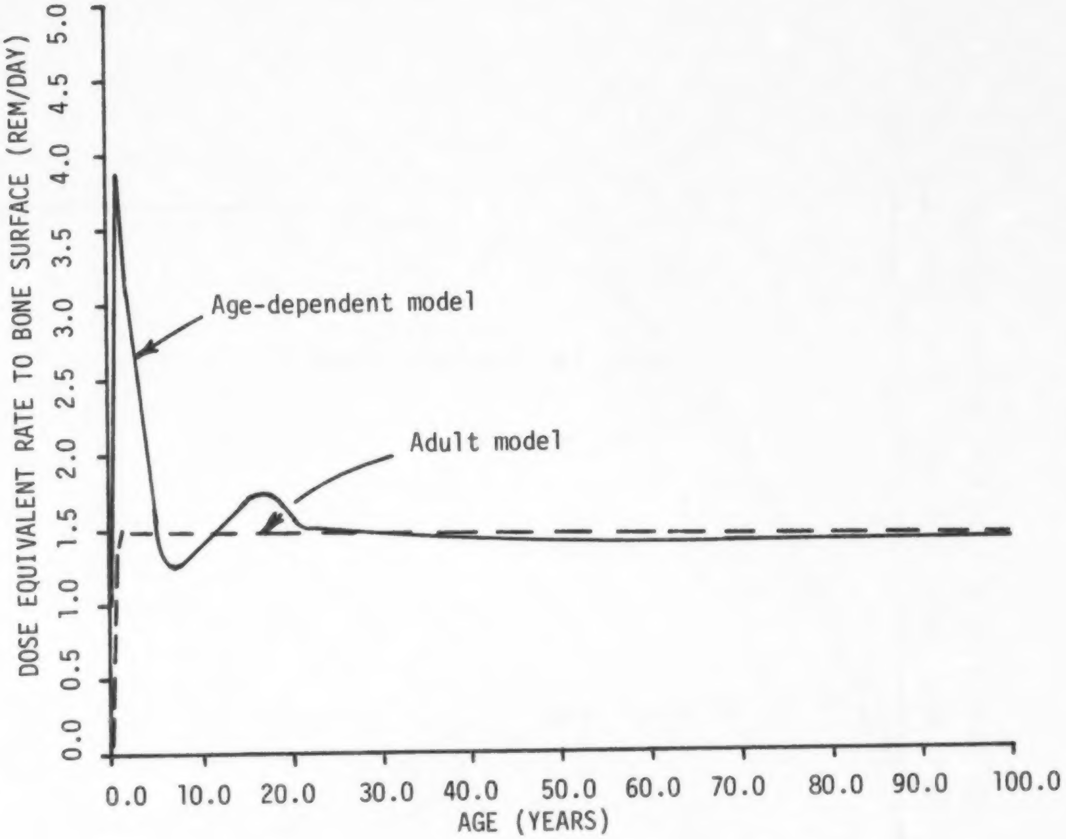


Figure F-7. Dose rate from chronic ingestion of strontium-90 in water at a concentration of 1 μCi/l

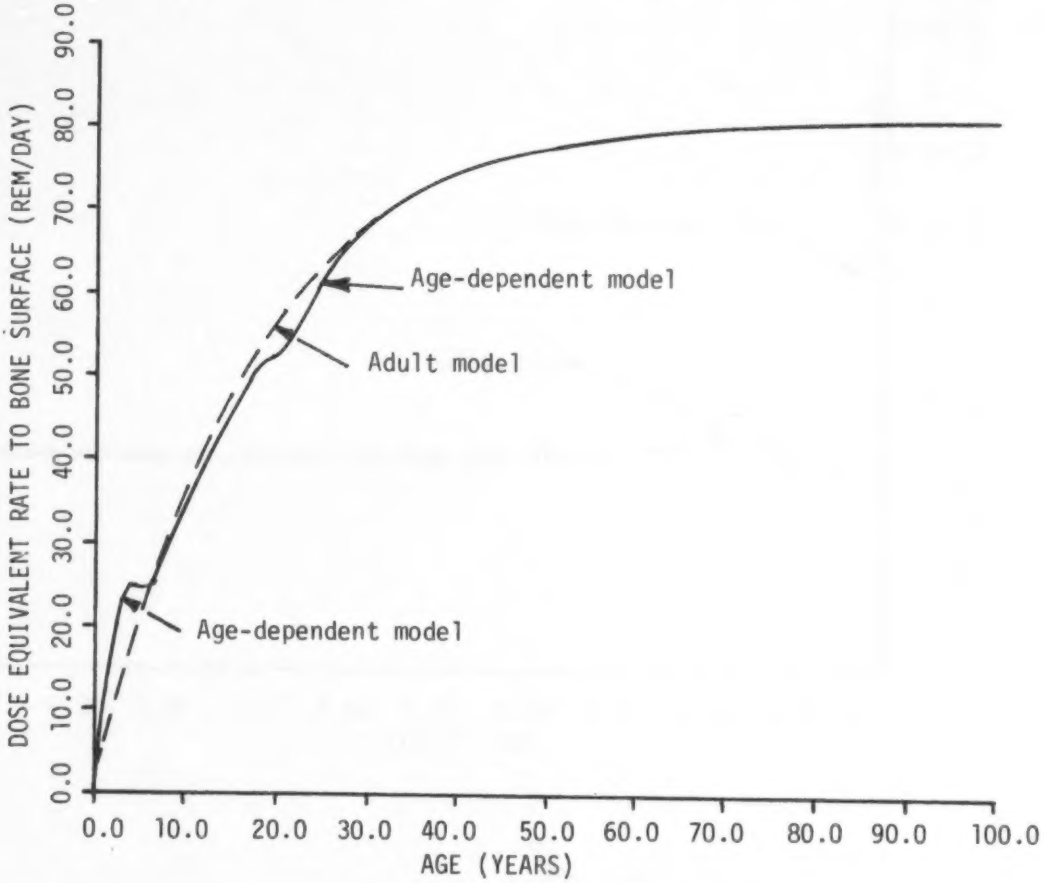


Figure F-8. Dose rate from chronic inhalation of strontium-90 in air at a concentration of 1 μCi/m3 trace the iron pathway, with the result that a substantial fraction of systemic plutonium is carried to the bone marrow and to the liver. In the skeleton, plutonium may be released mainly at sites of developing red cells. Plutonium that has reached the skeleton behaves very differently from iron; its movement is governed by fairly complicated processes of bone resorption and addition. Because the total metabolic behavior of plutonium is not closely related to that of any essential element, any retention model for plutonium as a function of age will involve much larger uncertainties than the analogous model for strontium. Still, there is enough information concerning the metabolism of plutonium by mammals to justify an examination of potential differences with age in doses to radiosensitive tissues following intake of this radioelement.

The effect of age-dependent parameters on dose rate calculations is most evident for the lung when the inhalation pathway is considered. Figure F-9 exhibits the variation in dose rate to the total and pulmonary portions of the lung both for the adult and age-dependent cases. The increased dose rate from age 0 to about 20 is typically caused by variations in the breathing rate-lung mass ratio for infants and juveniles. For this model, the age-dependent pulmonary lung 70-year dose is about 9 percent greater than for the adult model.

To describe retention of plutonium in the skeleton, it is convenient to view the skeleton as consisting of a cortical compartment and trabecular compartment. Each of these is further divided into three subcompartments: bone surface, bone volume, and a transfer compartment. The transfer compartment, which includes the bone marrow, may receive plutonium that is removed from bone surface or volume; plutonium may reside in this compartment temporarily before being returned either to the bloodstream or to bone surfaces (Figure F-10). Because of the large amount of recycling of plutonium among the skeletal compartments, blood, and other organs, recycling is considered explicitly in the model. The age-dependent features of the model are described in detail in (ORNL84a).

Red bone marrow dose rates for the age-dependent model are shown in Figure F-11, for ingestion, and in Figure F-12, for inhalation. The dashed curves are the dose rates using non-age-dependent parameters. As in the corresponding curves for strontium, the difference is more pronounced for the ingestion pathway. Because of the long physical and biological half-lives of plutonium in the skeleton, the dose rate, for a chronic intake, does not reach equilibrium within the one hundred year time period of the figures. The total lifetime (70-year) dose to the red marrow is about 25 percent greater for ingestion, and nearly unchanged for inhalation when the age-dependent parameters are used.

In summary, it is difficult to make generalizations concerning the uncertainty involved in neglecting age dependence in the dose calculations. Although the examples given indicate higher dose rates for the ingestion pathway, with smaller changes for inhalation, when using age-dependent parameters, this results from the complex interaction between parameters in the dose equation and depends on the element/organ combination under consideration.

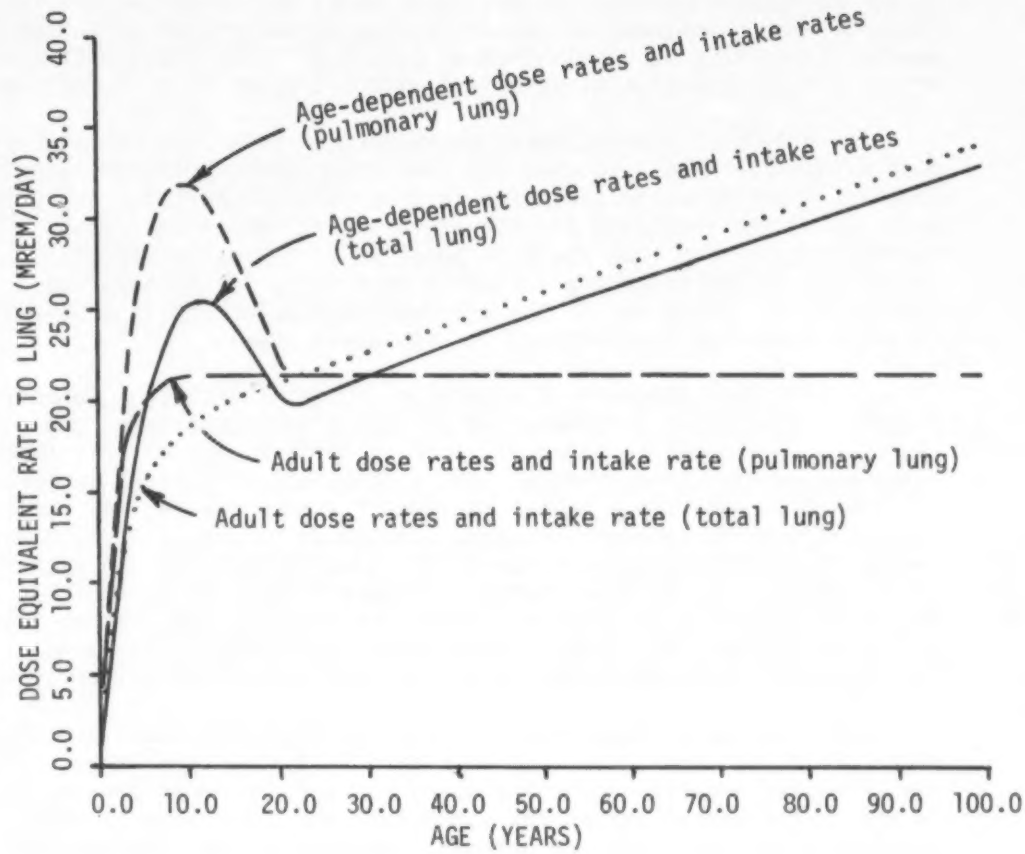
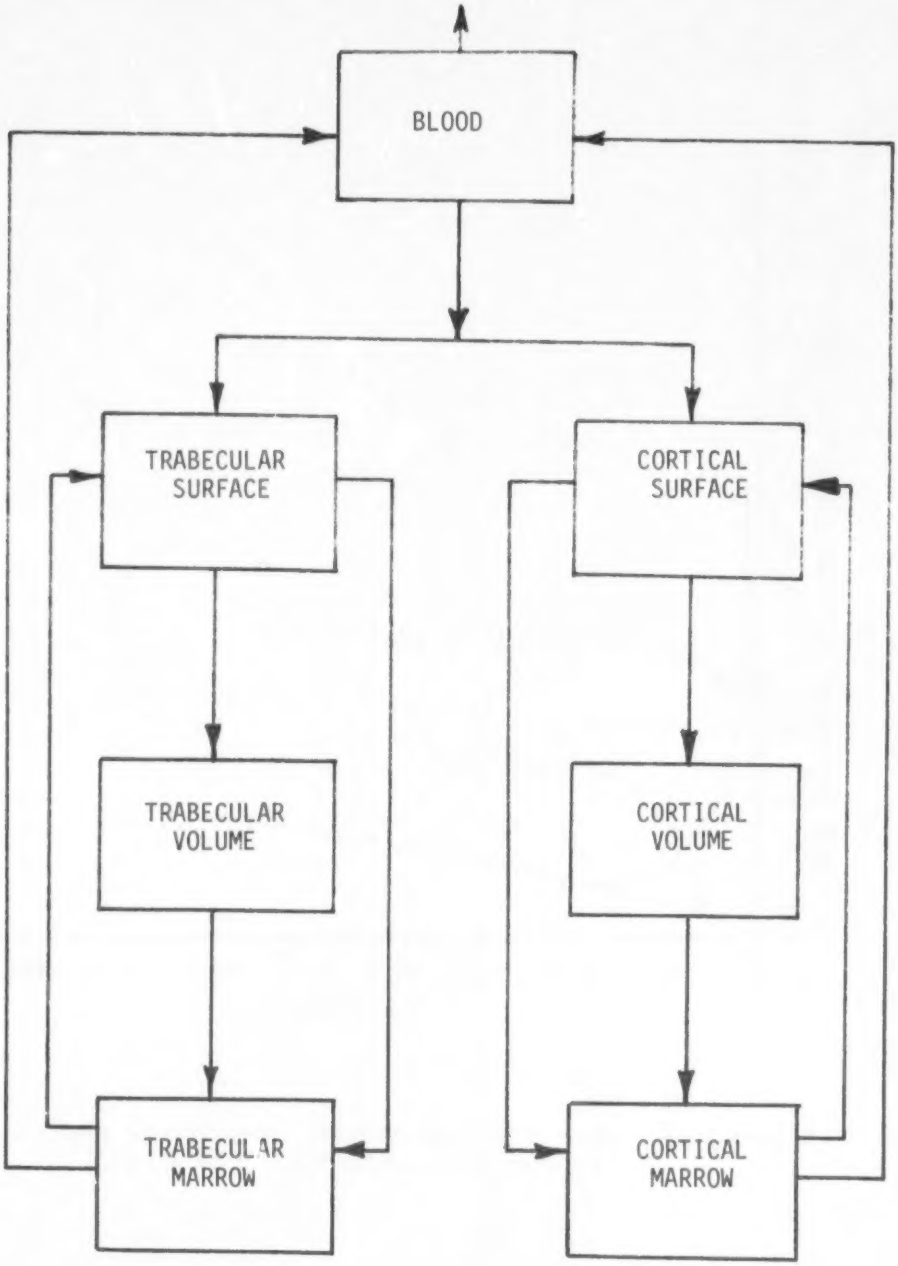


Figure F-9. Dose rate from chronic inhalation of plutonium-239 in air at a concentration of 1 μCi/m^3

Figure F-10. Compartments and pathways in model for plutonium in skeleton


BLOOD
TRABECULAR SURFACE
CORTICAL SURFACE
TRABECULAR VOLUME
CORTICAL VOLUME
TRABECULAR MARROW
CORTICAL MARROW

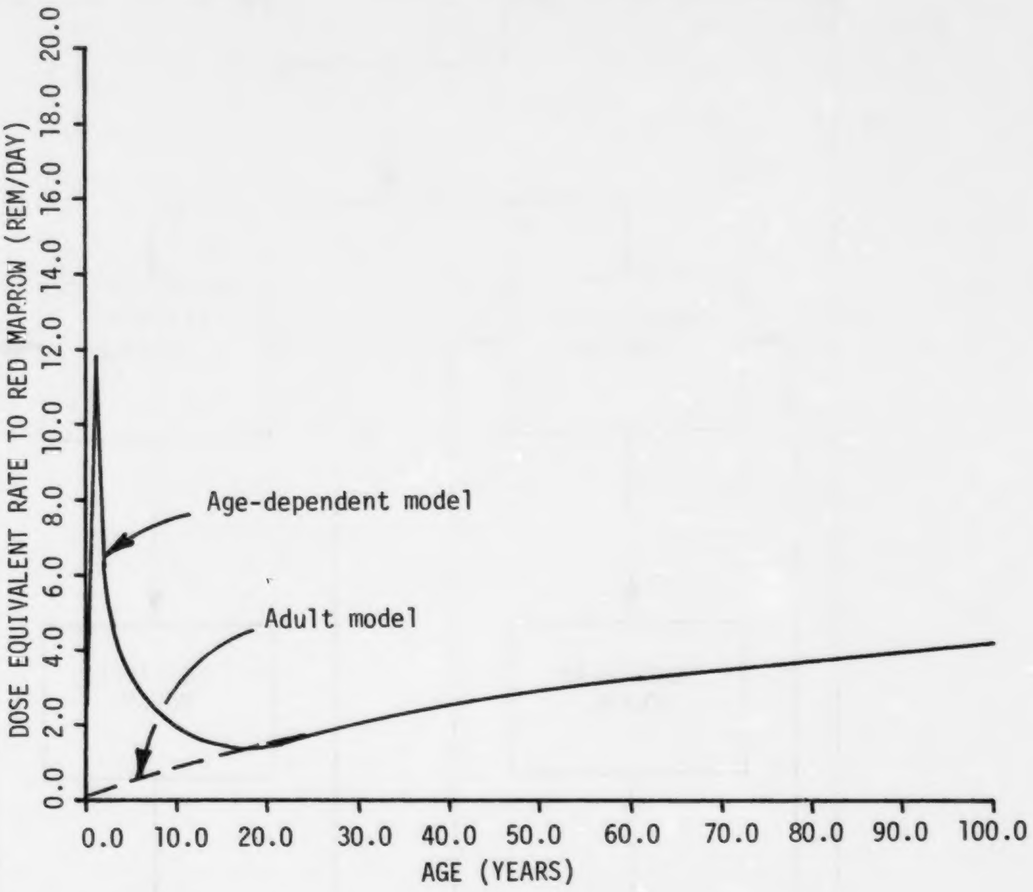


Figure F-11. Dose rate from chronic ingestion of plutonium-239 in water at a concentration of 1 μCi/l

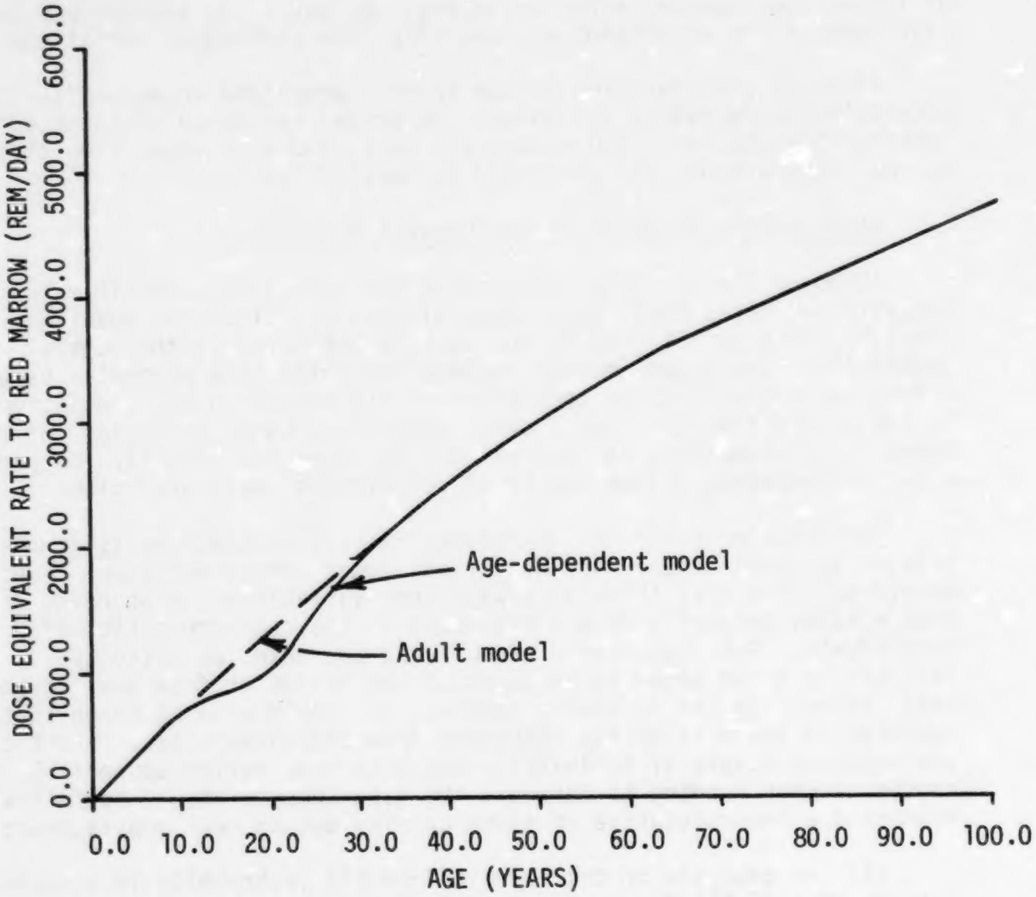


Figure F-12. Dose rate from chronic inhalation of plutonium-239 in air at a concentration of 1 $\mu Ci/m^3$

F.5.3 Dose Uncertainty Caused by Measurement Errors

The last potential source of uncertainty in the dose calculations is the error involved in making measurements of fixed quantities. The radioactive half-life of an isotope, for example, may be measured independently of any biological system, but the measurement is subject to some error. The organ mass of a given organ may also be measured with only a small error. Repeated determinations of these quantities, in addition, can reduce the error. Although this source of uncertainty may be of importance in other aspects of an environmental assessment, it is of little consequence in the dosimetry, because it is overwhelmed by the magnitude of the uncertainties resulting from individual variations.

Although consideration of the factors described above implies large uncertainties in calculated doses, the actual variation is expected to be considerably smaller. The reason for this, and some supporting studies on real populations, are presented in Section F.6.

F.6 Distribution of Doses in the General Population

Although the use of extreme parameter values in a sensitivity analysis indicates that large uncertainties in calculated doses are possible, this uncertainty is not usually reflected in the general population. There are several reasons for this: the parameter values chosen are intended to be typical of an individual in the population; it is improbable that the "worst case" parameters would be chosen for all terms in the equation; and not all of the terms are mutually independent, e.g., an increased intake may be offset by more rapid excretion.

This smaller range of uncertainty in real populations is demonstrated by studies performed on various human and animal populations. It should be noted that there is always some variability in observed doses that results primarily from differences in the characteristics of individuals. The usual way of specifying the dose, or activity, variability in an organ is in terms of the deviation from the average, or mean, value. In the following studies, it should also be noted that, in addition to the variability resulting from individual characteristics, the exposure levels of individuals may also have varied appreciably - another factor tending to increase the dose uncertainty. The following studies are representative of those carried out on real populations:

(1) An analysis of the thyroid from 133 jackrabbits in a nuclear fallout area (Tu65) found that in only 2 did the iodine-131 content exceed three times the same mean.

(2) Measurements of the strontium-90 content of adult whole skeletons (Ku62) showed that only about 5 percent of the population would exceed twice the average activity, with only about 0.1 percent exceeding four times the average.

(3) In another study, the cesium-137 content of 878 skeletal muscle samples (E164a, E164b) was measured. This radioisotope is also the result of nuclear tests so that the muscle content depends not only on the variation in individual parameters but also on the pathways leading to ingestion or inhalation of the isotope. Nevertheless, analyses of these samples indicated that only 0.2 percent exceeded three times the mean activity at a 95 percent confidence level.

(4) A study of the variability in organ deposition among individuals exposed under relatively similar conditions to toxic substances has also been performed (Cu79). In eleven exposure situations (Table F-3), the geometric standard deviation of the apparently lognormal organ doses ranged from 1.3 to 3.4. This means that 68 percent of the bone doses resulting from ingestion of strontium-90 would lie between 0.56 and 1.8 times the average.

In all but two of the situations examined, there is the complicating factor that there was probably a great deal of variation in the exposure levels experienced by members of the population. The magnitude of geometric standard deviations of the studies listed in Table F-3 may be the evidence of this variation since, except for the two beagle studies, the exposure was not uniform. Despite these nonuniform exposures, however, the organ dose is not greatly affected probably because of differences in metabolic processes. For example, there is probably some "self-adjustment" in the amount of strontium-90 absorbed from the small intestine to blood of different persons, since strontium-90 tends to vary with calcium in food; if a person has a low calcium intake, then he may absorb a higher fraction of the calcium and strontium-90 than a person with a high calcium intake.

In the beagle studies, the geometric standard deviation is 1.8 for inhaled metals in bone or liver, but is only 1.3 for ingested strontium-90 in bone. An important difference is that all dogs ingesting strontium-90 at a given level were administered the same amount, whereas, in the inhalation studies, the exposure air concentrations were controlled but the dogs inhaled variable amounts depending upon their individual characteristic breathing patterns.

Thus, in real situations, the overall uncertainty in dose is seen to be considerably smaller than would be expected solely on a basis of the "worst case" sensitivity analyses.

F.7 Summary

This appendix presents an overview of the methods used by EPA to estimate radiation doses. The appendix defines the basic quantities reported by EPA and describes briefly the models employed. The appendix also points out departures from the occupational parameters and assumptions employed in the basic ICRP methodology and gives the reasons for the deviations outlined.

TABLE F-3:
Distributions of organ doses[a] from inhalation and ingestion of metals

Population	Exposure	Principal exposure mode	Target organ	Geometric standard deviation of organ doses[a]
Beagle	Metals	Inhalation	Bone or liver	1.8
Humans	Plutonium (fallout)	Inhalation	Lung	3[b]
Humans	Titanium (soil)	Inhalation	Lung	3.4[b]
Humans	Aluminum (soil)	Inhalation	Lung	3.4[b]
Humans	Vanadium (fuel combustion)	Inhalation	Lung	3.4[b]
Beagles	Strontium-90	Ingestion	Bone	1.3
Humans	Strontium-90 (fallout)	Ingestion	Bone	1.8[b]
Humans (smokers)	Cadmium	Inhalation and Ingestion	Kidney	1.8[b]
Humans (nonsmokers)	Cadmium	Inhalation and Ingestion	Kidney	1.8[b]
Humans	Lead	Inhalation and Ingestion	Bone	2.2[b]
Humans	Lead	Inhalation	Lung	1.7[b]

[a] The stable element organ doses used in compiling this table were generally expressed in parts-per-million of organ mass.

[b] Note that exposure levels may vary considerably among individuals; if this factor could be eliminated, geometric standard deviations probably would be smaller.

Source: (Cu79).

Many of the physiological and metabolic parameters recommended in methods for calculating radiation doses are based on a limited number of observations, often on atypical humans or on other species. EPA has attempted to bound the uncertainty associated with the ranges observed for some of the more important parameters used. In fact, some empirical data on population doses mentioned here indicate that actual dose uncertainties are much less than is implied by this "worst case" analysis. For the sources of uncertainty discussed, the large dose ranges possible because of variation in individual characteristics must be modified by consideration of the narrower ranges indicated by studies of real populations; the dose range resulting from age dependence appears to be small for lifetime exposures, and the range resulting from experimental error is negligible by comparison. Based on these observations, it is reasonable to estimate that EPA's calculated doses should be accurate within a factor of 3 or 4. It should be emphasized that much of the "uncertainty" in the dose calculation is not caused by parameter error but reflects real differences in individual characteristics within the general population. Therefore, the uncertainty in the dose estimates cannot be dissociated from specification of the segment of the population to be protected.

More complete derivations and explanations for the EPA methodology are given in the references cited in the text, and a technical description of the dose rate equations and their use in conjunction with the life table risk evaluation is given in Appendix H.

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APPENDIX G: ESTIMATING THE RISK OF HEALTH EFFECTS RESULTING FROM RADIONUCLIDE RELEASES

G.1 Introduction

This appendix describes how EPA estimates the probability of fatal cancer, serious genetic effects, and other detrimental health effects caused by exposure to ionizing radiation. Such risk estimates are complex and uncertain, even though much scientific effort has been expended to increase the understanding of radiation effects.

Because the effects of radiation on human health are known more quantitatively than for most other environmental pollutants, it is possible to make numerical estimates of the risk from a particular source of radioactivity. Such numbers may give an unwarranted aura of certainty to estimated radiation risks. Compared to the baseline incidence of cancer and genetic defects, radiogenic cancer and radiation-induced genetic defects do not occur very frequently. Even among heavily irradiated populations, the number of cancers and genetic defects resulting from radiation is not known with either accuracy or precision simply because of sampling variability. In addition, exposed populations have not been followed for their full lifetime, so that information on ultimate effects is limited. Moreover, when considered in light of information gained from experiments with animals and from various theories of carcinogenesis and mutagenesis, the observational data on the effects of human exposure are subject to a number of interpretations. This in turn leads to differing estimates of radiation risks by both individual radiation scientists and expert groups. Readers should bear in mind that estimating radiation risks is not a mature science and that the evaluation of radiation hazards will change as additional information becomes available. In this appendix, a number of simple mathematical models are presented that may describe the main features of the human response to radiation. However, most scientists would agree that the underlying reality is quite complicated and largely unknown, so that such models should not be taken too literally but rather as useful approximations that will someday be obsolete.

EPA's estimates of cancer and genetic risks in this report are based on the 1980 National Academy of Sciences BEIR-3 report (NAS80). This report was prepared for the purpose of assessing radiation risks at the low exposure levels of interest in standard setting. As phrased by the President of the Academy, "We believe that the report will be helpful to the EPA and other agencies as they reassess radiation protection standards. It provides the scientific bases upon which standards may be decided after non- scientific social values have been taken into account."

In the sections below, we outline the various assumptions made in calculating radiation risks based on the 1980 NAS report and compare these risk estimates with those prepared by other scientific groups, such as the 1972 NAS BEIR Committee (NAS72), the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and the International Commission on Radiation Protection (ICRP). We recognize that information on radiation risks is incomplete and do not argue that the estimates made by the 1980 NAS BEIR Committee are highly accurate. Rather, we discuss some of the deficiencies in the available data base and point out possible sources of bias in current risk estimates. Nevertheless, we believe the risk estimates made by EPA are "state-of-the-art."

In the sections below, we first consider the cancer risk resulting from whole-body exposure to low-LET* radiation, i.e., lightly ionizing radiation like the energetic electrons produced by X-rays or gamma rays. Environmental contamination by radioactive materials also leads to the ingestion or inhalation of the material and subsequent concentration of the radioactivity in selected body organs. Therefore, the cancer risk resulting from low-LET irradiation of specific organs is examined next. Organ doses can also result from high-LET radiation, such as that associated with alpha particles. The estimation of cancer risks for situations where high-LET radiation is distributed more or less uniformly within a body organ is the third situation considered, Section G.3. In Section G.4, we review the causes of uncertainty in the cancer risk estimates and the magnitude of this uncertainty so that the public as well as EPA decision makers have a proper understanding of the degree of confidence to place in them. In Section G.5, we review and quantify the hazard of deleterious genetic effects from radiation and the effects of exposure *in utero* on the developing fetus. Finally, in Section G.6, we calculate cancer and genetic risks from background radiation using the models described in this appendix.

G.2 Cancer Risk Estimates for Low-LET Radiations

Most of the observations of radiation-induced carcinogenesis in humans are on groups exposed to low-LET radiations. These groups include the Japanese A-bomb survivors and medical patients treated with X-rays for ankylosing spondylitis in England from 1935 to 1954 (Sm78). The UNSCEAR (UNSCEAR77) and NAS Committee on the Biological Effects of Ionizing Radiations (BEIR) (NAS80) have provided knowledgeable reviews of these and other data on the carcinogenic effects of human exposures.

The most important epidemiological data base on radiogenic cancer is the A-bomb survivors. The Japanese A-bomb survivors have been studied for more than 38 years and most of them, the Life Span Study Sample, have been followed in a carefully planned and monitored epidemiological survey since 1950 (Ka82, Wa83). They were exposed to a wide range of doses and are the largest group that has been studied.

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*Linear Energy Transfer (LET) -- the energy deposited per unit of distance along the path of a charged particle.

Therefore, they are virtually the only group providing information on the response pattern at various levels of exposure to low-LET radiation. Unfortunately, the doses received by various individuals in the Life Span Study Sample are not yet known accurately. The 1980 BEIR Committee's analysis of the A-bomb survivor data was prepared before bias in the dose estimates for the A-bomb survivors (the tentative 1965 dose estimates, T65) became widely recognized (Lo81). It is now clear that the T65 doses tended to be overestimated (Bo82, RERF83,84) so that the BEIR Committee's estimates of the risk per unit dose are likely to be too low. A detailed reevaluation of current risk estimates is indicated when the A-bomb survivor data have been reanalyzed on the basis of new and better estimates of the dose to individual survivors.

Uncertainties in radiation risk estimates do not result just from the uncertainties in the Japanese data base and in other epidemiological studies. Analyses of these data bases require a number of assumptions that have a considerable effect on the estimated risk. These assumptions are discussed below. The degree of uncertainty introduced by choosing among these assumptions is probably greater than the uncertainty of the estimated risk per unit dose among the A-bomb survivors or other sources of risk estimates for radiogenic cancer in humans.

G.2.1 Assumptions Needed to Make Risk Estimates

A number of assumptions must be made about how observations at high doses should be applied at low doses and low dose rates for radiation of a given type (LET). These assumptions include the shape of the dose response function and possible dose rate effects. A dose response function expresses the relationship between dose and the probability that a radiogenic cancer is induced. Observed excess cancers have occurred, for the most part, following relatively high doses of ionizing radiation compared to those likely to occur as a result of the combination of background radiation and environmental contamination from controllable sources of radiation. Therefore, a dose response model provides a method of interpolating between the number of radiogenic cancers observed at high doses and the number of cancers resulting from all causes including background radiation.

The range of interpolation is not the same for all kinds of cancer because it depends upon the radiosensitivity of a given tissue. For example, the most probable radiogenic cancer for women is breast cancer. As described below, with appropriate references, breast cancer appears not to be reduced when the dose is delivered over a long period of time. For example, the number of excess cancers per unit dose among Japanese women, who received acute doses, is about the same per unit dose as women exposed to small periodic doses of X-rays over many years. If this is actually the case, background radiation is as carcinogenic for breast tissue as the acute exposures from A-bomb gamma radiation. Moreover, the female A-bomb survivors show an excess of breast cancer at doses below 20 rad which is linearly proportional to that observed at several hundred rad (To84). Women in their 40's, the youngest age group in which breast cancer is common, have received about 4 rad of whole-body low-LET background radiation and usually some additional dose incurred for diagnostic medical purposes. Therefore, for this cancer, the difference between observed radiogenic cancer, less than 20 rad, and the dose resulting from background radiation is less than a factor of 5, not several orders of magnitude as is sometimes claimed. However, it should be noted that breast tissue is a comparatively sensitive tissue for cancer induction and that for most cancers, a statistically significant excess has not been observed at doses below 100 rad, low-LET. Therefore, the range of dose interpolation between observed and calculated risk is often large.

G.2.2 Dose Response Functions

The 1980 NAS report (NAS80) examined three dose response functions in detail: (1) linear, in which effects are directly proportional to dose at all doses; (2) linear quadratic, in which effects are very nearly proportional to dose at very low doses and proportional to the square of the dose at high doses; and (3) a quadratic dose response function, where the risk varies as the square of the dose at all dose levels.

We believe the first two of these functions are compatible with most of the data on human cancer. Information which became available only after the BEIR-3 report was published indicates that a quadratic response function is inconsistent with the observed excess risk of solid cancers at Nagasaki, where the estimated gamma-ray doses are not seriously confounded by an assumed neutron dose component. The chance that a quadratic response function underlies the excess cancer observed in the Nagasaki incidence data has been reported as only 1 in 10,000 (Wa83). Although a quadratic response function is not incompatible with the Life Span Study Sample data on leukemia incidence at Nagasaki, Beebe and others (Be78, El77) have pointed out how unrepresentative these data are of the total observed dose response for leukemia in that city. There is no evidence that a quadratic response function provides a better fit to the observed leukemia excess among all A-bomb survivors in the Life Span Study Sample than a simple linear model (NAS80). Based on these considerations, we do not believe a quadratic response can be used in a serious effort to estimate cancer risks due to ionizing radiation. EPA notes that neither the NCRP, the ICRP, nor other authoritative scientific groups, e.g., NRPB and UNSCEAR, have used a quadratic response function to estimate the risks due to ionizing radiation.

The 1980 NAS BEIR Committee considered only the Japanese mortality data in their analysis of possible dose response functions (NAS80). Based on the T65 dose estimates, this Committee showed that the excess incidence of solid cancer and leukemia among the A-bomb survivors is compatible with either a linear or linear quadratic dose response to the low-LET radiation component and a linear response to the high-LET neutron component (NAS80). Although the 1980 BEIR report indicated low-LET risk estimates based on a linear quadratic response were "preferred" by most of the scientists who prepared that report, opinion was not unanimous, and we believe the subsequent reassessment of the A-bomb dose seriously weakens the Committee's conclusion. The Committee's analysis of dose response functions was based on the assumption that most of the observed excess leukemia and solid cancers among A-bomb survivors resulted from neutrons (NAS80). Current evidence, however, is conclusive that neutrons were only a minor component of the dose in both Hiroshima and Nagasaki (Bo82, RERF83,84). Therefore, it is likely that the linear response attributed to neutrons was caused by the gamma dose, not the dose from neutrons. This point is discussed further in Section G.3.

Reanalysis of the Japanese experience after completion of the dose reassessment may provide more definitive information on the dose response of the A-bomb survivors, but it is unlikely to provide a consensus on the dose response at environmental levels, i.e., about 100 mrad per year. This is because at low enough doses there will always be sampling variations in the observed risks so that observations are compatible, in a statistical sense, with a variety of dose response functions. In the absence of empirical evidence or a strong theoretical basis, a choice between dose response functions must be based on other considerations.

Although there is evidence for a nonlinear response to low-LET radiations in some, but not all, studies of animal radiocarcinogenesis (see below), we are not aware of any data on human cancers that are incompatible with a simple linear model. In such a case, it may be preferable to adopt the simplest hypothesis that adequately models the observed radiation effect. Occams's razor is still a viable scientific rule for separating necessary from ad hoc assumptions. Moreover, EPA believes that risk estimates, for the purpose of assessing radiation impacts on public health, should be based on scientifically creditable risk models that are unlikely to understate the risk. The linear model fulfills this criteria. Given the current bias in the doses assigned to A-bomb survivors (see Section 7.5.1 below), such an approach seems reasonable, as well as prudent. Therefore, in this appendix, EPA has used the BEIR-3 linear dose response model as one of two dose response models for discussing the risk of radiogenic cancer due to low-LET radiations.

For low-LET radiations, we have also included in the appendix, discussions of risk that are based on the BEIR-3 linear quadratic dose response model. While in the dose range of interest (environmental levels) the dose squared term in this model is insignificant, the linear term is about 2.5 times smaller than that in the BEIR-3 linear response model, NAS80. That is, for the same dose, risk estimates based on the BEIR-3 linear quadratic dose response model are only 40 percent of those based on the BEIR-3 linear model.

Many of the risk estimates needed to evaluate the effect of radionuclide emission must be made on an organ specific basis. The BEIR-3 report provides risk coefficients for individual solid cancers only for the linear model, Tables V-14 and V-15 in NAS80. We have therefore divided BEIR-3 organ risk estimates for a linear response by a factor of 2.5 to obtain organ specific linear quadratic risk coefficients.

The underlying basis for a linear quadratic response is thought to be that repair of radiation damage mitigates the effect of small doses of radiation or those which occur over a long time period, the reduced linear term being indicative of this repair. Use of a linear quadratic dose response function, as formulated by the BEIR-3 Committee, is equivalent to the use of a dose rate effectiveness factor (DREF) of 2.5 (see below).

The discussions of both the linear and the linear-quadratic dose response models for low-LET radiations are included in this appendix to compare the risk estimates obtained for given doses using both models. The more conservative of these two models is the linear model. We have used this model for the calculation of the fatal cancers per curie release to the accessible environment which are listed in Chapter 6. This policy was thoroughly reviewed and accepted by the High-Level Radioactive Waste Disposal Subcommittee of the EPA Science Advisory Board (EPA84).

G.2.3 The Possible Effects of Dose Rate on Radiocarcinogenesis

The BEIR-3 Committee limited its risk estimates to a minimum dose rate of 1 rem per year and stated that it "does not know if dose rates of gamma rays and X-rays of about 100 mrad/y are detrimental to man." At dose rates comparable to the annual dose that everyone receives for naturally-occurring radioactive materials, a considerable body of scientific opinion holds that the effects of radiation are reduced. NCRP Committee 40 has suggested that carcinogenic effects of low-LET radiations may be a factor of from 2 to 10 times less for small doses and dose rates than have been observed at high doses (NCRP80).

The low dose and low dose rate effectiveness factors developed by NCRP Committee 40 are based on their analysis of a large body of plant and animal data that showed reduced effects at low doses for a number of biological endpoints, including radiogenic cancer in animals, chiefly rodents. However, no data for cancer in humans confirm these findings as yet. A few human studies contradict them. Highly fractionated small doses to human breast tissue are apparently as carcinogenic as large acute doses (NAS80, La80). Furthermore, small acute (less than 10 rad) doses to the thyroid are as effective per rad as much larger doses in initiating thyroid cancer (UNSCEAR77, NAS80). Moreover, the increased breast cancer resulting from chronic low dose occupational gamma ray exposures among British dial painters is comparable to, or larger, than that expected on the basis of acute high dose exposures (Ba81).

While none of these examples is persuasive by itself, collectively they indicate that it may not be prudent to assume that all kinds of cancer are reduced at low dose rates and/or low doses. However, it may be overly conservative to estimate the risk of all cancers on the basis of the linearity observed for breast and thyroid cancer. The International Commission on Radiation Protection and the United Nations Scientific Committee on Atomic Radiations have used a dose rate effectiveness factor of about 2.5 to estimate the risks from occupational (ICRP-26) and environmental exposures (UNSCEAR77). Their choice of a DREF is fully consistent with and equivalent to the reduction of risk at low doses obtained by substituting the BEIR-3 linear-quadratic response model for their linear model. Use of both a DREF and a linear quadratic model for risk estimation is inappropriate (NCRP80).

The difference between risk estimates obtained with the BEIR-3 linear and linear-quadratic dose response models is by no means the full measure of the uncertainty in the estimates of the cancer risk resulting from ionizing radiation (Section G.4 below summarizes information on uncertainty). Using two models serves as a reminder that there is more than one creditable dose response model for estimating radiation risks and that it is not known if all radiogenic cancers have the same dose response.

G.2.4 Risk Projection Models

None of the exposed groups have been observed long enough to assess the full effects of their exposures, if, as currently thought, most radiogenic cancers occur throughout an exposed person's lifetime (NAS80). Therefore, another major decision that must be made in assessing the lifetime cancer risk due to radiation is to select a risk projection model to estimate the risk for a longer period of time than currently available observation data will allow.

To estimate the risk of radiation exposure that is beyond the years of observation, either a relative risk or an absolute risk projection model (or suitable variations) may be used. These models are described at length in Chapter 4 of the 1980 NAS report (NAS80). A relative risk projection model projects the currently observed percentage increase in cancer risk per unit dose into future years. An absolute risk model projects the average observed number of excess cancers per unit dose into future years at risk.

Because the underlying risk of cancer increases rapidly with age, the relative risk model predicts a larger probability of excess cancer toward the end of a person's lifetime. In contrast, the absolute risk model predicts a constant incidence of excess cancer across time. Therefore, given the incomplete data we have now, less than lifetime follow-up, a relative risk model projects somewhat greater risk than that projected using an absolute risk model.

The National Academy of Sciences BEIR Committee and other scientific groups, e.g., UNSCEAR, have not concluded which projection model is the appropriate choice for most radiogenic cancers. However, evidence is accumulating which favors the relative risk projection model for most solid cancers. As pointed out by the 1980 NAS BEIR Committee,

> "If the relative-risk model applies, then the age of the exposed groups, both at the time of exposure and as they move through life, becomes very important. There is now considerable evidence in nearly all the adult human populations studied that persons irradiated at higher ages have, in general, a greater excess risk of cancer than those irradiated at lower ages, or at least they develop cancer sooner. Furthermore, if they are irradiated at a particular age, the excess risk tends to rise pari passu (at equal pace) with the risk of the population at large. In other words, the relative-risk model with respect to cancer susceptibility at least as a function of age, evidently applies to some kinds of cancer that have been observed to result from radiation exposure." (NAS80, p.33)

This observation is confirmed by the Ninth A-bomb Survivor Life Span Study, published two years after the 1980 Academy report. This latest report indicates that, for solid cancers, relative risks have continued to remain constant in recent years while absolute risks have increased substantially (Ka82). Smith and Doll (Sm78) have reached similar conclusions on the trend in excess cancer with time among the irradiated spondylitic patients.

Although we believe considerable weight should be given to the relative risk model for most solid cancers (see below), the model does not necessarily give an accurate projection of lifetime risk. The mix of tumor types varies with age so that the relative frequency of some common radiogenic tumors, such as thyroid cancer, decreases for older ages. Land has pointed out that this may result in overestimates of the lifetime risk when they are based on a projection model using summed sites, relative risks (La83). While this may turn out to be true for estimates of cancer incidence that include cancers less likely to be fatal, e.g., thyroid, it may not be too important in estimating the lifetime risk of fatal cancers since the incidence of most of the common fatal cancers, e.g., breast and lung cancers, increases with age.

Leukemia and bone cancer are exceptions to the general validity of a lifetime expression period for radiogenic cancers. Most, if not all, of the leukemia risk has apparently already been expressed in both the A-bomb survivors and the spondylitics (Ka82, Sm78). Similarly, bone sarcoma from acute exposure appears to have a limited expression period (NAS80, Ma83). For these diseases, the BEIR-3 Committee believed that an absolute risk projection model with a limited expression period is appropriate for estimating lifetime risk (NAS80).

Note that, unlike the NAS BEIR-1 report (NAS72), the BEIR-3 Committee's relative and absolute risk models are age dependent. That is, the risk coefficient changes, depending on the age of the exposed persons. Observation data on how cancer risk resulting from radiation changes with age is sparse, particularly so in the case of childhood exposures. Nevertheless, the explicit consideration of the variation in radiosensitivity with age at exposure is a significant improvement in methodology. It is important to differentiate between age sensitivity at exposure and the age dependence of cancer expression. In general, people are most sensitive to radiation when they are young. In contrast most radiogenic cancers occur late in life, much like cancers resulting from other causes. In this appendix we present risk estimates for a lifetime exposure of equal annual doses. The cancer risk estimated is lifetime risk from this exposure pattern. However, age-dependent analyses using BEIR-3 risk coefficients indicate that the risk from one year of exposure varies by a factor of at least 5, depending on the age of the recipient.

G.2.5 Effect of Various Assumptions on the Numerical Risk Estimates

Differences between risk estimates made by using various combinations of the assumptions described above were examined in the 1980 NAS report. Table G-1 below, taken from Table V-25 (NAS80), shows the range of cancer fatalities that are induced by a single 10-rad dose as estimated using linear, linear quadratic, and quadratic dose response functions and two risk projection models, relative and absolute (NAS80).

As illustrated in Table G-1, estimating the cancer risk for a given risk projection model on the basis of a quadratic as compared to a linear dose response reduces the estimated risk of fatal cancer by a factor of nearly 20. Between the more credible linear and linear quadratic response functions, the difference is less, a factor of about 2.5. For a given dose response model, results obtained with the two projection models, for solid cancers, differ by about a factor of 3.

Even though the 1980 NAS analysis estimated lower risks for a linear quadratic response, it should not be concluded that this response function always provides smaller risk estimates. In contrast to the 1980 NAS analysis where the proportion of risk due to the dose squared term (e.g., C_3 in equation c of Table G-1) was constrained to positive values, the linear quadratic function (which agrees best with Nagasaki cancer incidence data) has a negative coefficient for the dose-squared term (Wa83). Although this negative coefficient is small and indeed may not be significant, the computational result is a larger linear term which leads to higher risk estimates at low doses than would be estimated using a simple linear model (Wa83). Preliminarily, the BEIR-3 analyses of the mortality, which were not restricted to positive coefficients of the dose squared terms, yielded similar results.

Differences in the estimated cancer risk introduced by the choice of the risk projection model are also appreciable. As pointed out above, the 1980 NAS analysis indicates that relative risk estimates exceed absolute risk estimates by about a factor of 3, Table G-1. However, relative risk estimates are quite sensitive to how the risk resulting from exposure during childhood persists throughout life. This question is addressed in the next section, where we compare risk estimates made by the 1972 and 1980 NAS BEIR Committees with those of the ICRP and UNSCEAR.

TABLE G-1:
Range of cancer fatalities induced by 10-rad low-LET radiation
(Average value per rad per million persons exposed)

Dose response functions	Lifetime risk projection model	
	Relative[(a)]	Absolute
Linear[(b)]	501	167
Linear Quadratic[(c)]	226	77
Quadratic[(d)]	28	10

[(a)] Relative risk projection for all solid cancers except leukemia and bone cancer fatalities, which are projected by means of the absolute risk model (NAS80).

[(b)] Response R varies as a constant times the dose, i.e., $R=C_1D$.

[(c)] $R=C_2D+C_3D^2$.

[(d)] $R=C_4D^2$.

Source: NAS80, Table V-25.

G.2.6 Comparison of Cancer Risk Estimates for Low-LET Radiation

A number of estimates of the risk of fatal cancer following lifetime exposure are compared in Table G-2. Although all of these risk estimates assume a linear response function, they differ considerably because of other assumptions. In contrast with absolute risk estimates, which have increased since the first NAS report (BEIR-1) was prepared in 1972 (NAS72), the 1980 NAS BEIR-3 Committee's estimates of the relative risk, as shown in Table G-2, have decreased relative to those in the BEIR-1 report. This illustrates the sensitivity of risk projections to changes in modeling assumptions. For the NAS80 report, the relative risk observed for ages 10 to 19 was substituted for the considerably higher relative risk observed for those exposed during childhood, ages 0 to 9. In addition, the relative risk coefficients used in the BEIR-3 analysis are based on excess cancer in the Japanese A-bomb survivors compared to U.S. population cancer mortality rates. In the 1972 NAS report this excess was compared to cancer mortality in Japan. Moreover, the difference introduced by these two changes, particularly the former, is somewhat greater than indicated in the 1980 NAS report. The relative risk estimate attributed to the BEIR-1 Committee in the NAS 1980 report is incorrect. Therefore, two BEIR-1 relative risk estimates are listed in Table G-2: the risk estimate in NAS80 attributed to the BEIR-1 Committee and an estimate which is based on the risk coefficients in NAS72. The NAS 1980 estimate did not use the relative risk coefficient for childhood exposure given in the BEIR-1 report, which for solid cancers is a factor of 10 larger than adult values (p. 171 in NAS72), but rather used the adult risk for all ages including children. The estimate in Table G-2 labeled NAS72 uses the relative risk coefficients actually given in the BEIR-1 report.

By comparing the three relative risk estimates in Table G-2, it is apparent that the relative risk estimates are fairly sensitive to the assumptions made as to what extent the observed high relative risk of cancer from childhood exposure continues throughout adult life. The Life Span Study (Ka82) indicates that the high-risk adult cancer caused by childhood exposures is continuing, although, perhaps, not to the extent predicted by the NAS BEIR-1 Committee in 1972.

The major reason the risk estimates in Table G-2 differ is because of the underlying assumption in each set of risk estimates. The NAS BEIR estimates are for lifetime exposure and lifetime expression of induced cancers (NAS72, 80). Neither the age distribution of the population at risk nor the projection models (if any) have been specified by either the UNSCEAR (UNSCEAR77) or the ICRP (ICRP77). UNSCEAR apparently presumes the same age distributions as occurred in the epidemiological studies they cited, mainly the A-bomb survivors, and a 40-year period of cancer expression. The ICRP risk estimates are for adult workers, presumably exposed between ages 18 and 65, and a similar expression period. These are essentially age-independent absolute risk models with less than lifetime expression of induced cancer mortality. For these reasons alone, risks estimated by ICRP and UNSCEAR are expected to be smaller than those made on the basis of the BEIR-3 report.

The last entry in Table G-2 (Ch83) is of interest because it specifically excludes the A-bomb survivor data based on T65 dose estimates. The authors reanalyzed the information on radiogenic cancer in UNSCEAR77 so as to exclude all data based on the Japanese experience. Their estimate of fatalities ranges from 100 to 440 per

TABLE G-2:
A comparison of estimates of the risk of fatal cancer from a lifetime exposure at 1 rad/year (low-LET radiation)

Source of estimate	Cases per 10^6 person rad	Projection model
BEIR-1 (NAS72)(a)	667	Relative Risk
BEIR-1 (NAS80)(b)	568	Relative Risk
BEIR-3 (NAS80)(b)(c)	403	Relative Risk
BEIR-3 (NAS80)(d)	169	Relative Risk
BEIR-3 (NAS80)(b)	158	Absolute Risk
BEIR-1 (NAS80)(b)	115	Absolute Risk
BEIR-3 (NAS80)(d)	67	Absolute Risk
UNSCEAR (UNSCEAR77)(e)	200-300	None-high dose > 100 rad
UNSCEAR (UNSCEAR77)(e)	75-175	None-low dose/dose rate
ICRP (ICRP77)	125	None-occupational - low dose/dose rate
CLM (Ch83)	100-440	UNSCEAR77 without A-bomb data

(a) BEIR-1 relative risk model.

(b) Table V-4 in NAS80, linear dose response.

(c) L-L absolute risk model for bone cancer and leukemia; L-L relative risk model for all other cancer.

(d) Table V-4 in NAS80 linear-quadratic dose response.

(e) Paragraphs 317 and 318 in UNSCEAR77.

10^6 person rad for high doses and dose rates. As indicated in Table G-2, this is somewhat greater but comparable to the UNSCEAR estimate, which includes the A-bomb survivor data. The mean number of fatalities given in Ch83 is 270 per 10^6 person-rem, which is nearly identical to the value EPA has used for a linear dose response model--280 fatalities per 10^6 person rad (see below).

G.2.7 EPA Assumptions About Cancer Risks Resulting from Low-LET Radiations

EPA's discussion of radiation risks in this appendix are based on presumed linear and linear quadratic dose response functions. We believe these are the most credible dose response functions for estimating risks to exposed populations. Using the BEIR-3 linear quadratic model is equivalent, at low dose, to using a dose rate effectiveness factor of

2.5. As discussed in section G.2.2, we have used a linear dose response function for low-LET radiation in computing the fatal cancers per curie released to the accessible environment which are listed in Tables 6-1 through 6-4.

Except for leukemia and bone cancer, where we use a 25-year expression period for radiogenic cancer, we use a lifetime expression period, as was done in the NAS report (NAS80). Because the most recent Life Span Study Report (Ka82) indicates absolute risks for solid cancers are continuing to increase 33 years after exposure, the 1980 NAS Committee choice of a lifetime expression period appears to be well founded. We do not believe limiting cancer expression to 40 years (as has been done by the ICRP and UNSCEAR) is compatible with the continuing increase in solid cancers that has occurred among irradiated populations (Ka82). Analyses of the spondylitic data have led others to similar conclusions (Sm78).

To project the number of fatalities resulting from leukemia and bone cancer, EPA uses an absolute risk model, a minimum induction period of 2 years, and a 25-year expression period. To estimate the number of fatalities resulting from other cancers, EPA uses the arithmetic average of absolute and relative risk projection models. For these cancers, we assume a 10-year minimum induction period and expression of radiation-induced cancer for the balance of an exposed person's lifetime after the minimum induction period.

G.2.8 Methodology for Assessing the Risk of Radiogenic Cancer

EPA uses a life table analysis to estimate the number of fatal radiogenic cancers in an exposed population of 100,000 persons. This analysis considers not only death due to radiogenic cancer, but also the probabilities of other competing causes of death which are, of course, much larger and vary considerably with age (Bu81, Co78). Basically, it calculates for ages 0 to 110 the risk of death due to all causes by applying the 1970 mortality data from the National Center for Health Statistics (NCHS75) to a cohort of 100,000 persons. Additional information on the details of the life table analysis is provided in Appendix H. It should be noted that a life table analysis is required to use the age-dependent risk coefficients in the BEIR-3 report. For relative risk estimates, we use age-specific cancer mortality data also provided by NCHS (NCHS73). The EPA computer program we use for the life table analysis was furnished to the NAS BEIR-3 Committee by EPA and used by the Committee to prepare its risk estimates. Therefore, we believe that the population base and calculational approach are similar in both the NAS and EPA analyses.

To project the observed risks of most solid radiogenic cancers beyond the period of current observation, we use both absolute and relative risk models, but usually present an arithmetic average based on these projections. Using a single estimate, instead of a range of values, does not mean that our estimate is precise. As indicated in

Table G-2, the range of estimated fatal cancers resulting from the choice of a particular projection model and its internal assumptions is about a factor of 3. Although we think it is likely that the relative risk model is the best projection model for most solid cancers, it has been tested rigorously only for lung and breast cancer (La78). Until it has more empirical support, we prefer to use an average risk based on both projection models. A second reason for this choice is to avoid overly conservative risk estimates caused by the compounding of multiplicative conservative assumptions.

To estimate the cancer risk from low-LET, whole-body, lifetime exposure with the linear model, we use the arithmetic average of relative and absolute risk projections (the BEIR-3 $\overline{\text{L-L}}$ model) for solid cancers and an absolute risk projection for leukemia and bone cancer (the BEIR-3 L-L model). For a dose to the whole-body, this yields an estimated 280 fatalities per million person rad. For the BEIR-3 linear quadratic model, which is equivalent to assuming a DREF of 2.5, a low-LET whole body dose yields an estimated life risk of about 110 fatalities per million person rad.

These risk estimates are not unduly conservative. More than 235 of the 280 fatalities estimated with the BEIR-3 linear model result from cancers in soft tissues for which we have used the BEIR-3 $\overline{\text{L-L}}$ model. As explained on page 187 of that report (NAS80), the $\overline{\text{L-L}}$ model is not derived from the observed risk of solid cancers alone but rather includes parameters based on the Committee's analysis of the leukemia mortality data. Therefore, as outlined in Section G.4, the BEIR-3 Committee's analysis of the Japanese leukemia data depended heavily on the assumption that most of the leukemia observed at Hiroshima was caused by neutrons. In contrast, Table V-30 in the BEIR-3 report (NAS80) estimates the risk of cancer incidence in soft tissues directly, without the additional assumptions contained in the BEIR-3 $\overline{\text{L-L}}$ model. By using the weighted incidence mortality ratios given in the Table V-15 (NAS80), the results given in Table V-30 can be expressed in terms of mortality to yield, for lifetime exposure, an absolute risk estimate of about 200 fatalities per 10^6 person rad and about 770 fatalities per 10^6 person rad when a relative risk projection model is used to estimate lifetime risk. The arithmetic mean of the fatalities projected by these two models is almost 500 per 10^6 person rad, more than twice as many fatal soft tissue cancers as predicted by the BEIR-3 $\overline{\text{L-L}}$ model and about 5 times as many as estimated using the BEIR-3 linear quadratic model.

By a whole-body dose, we mean a uniform dose to every organ in the body. In practice, such exposure situations seldom occur, particularly for ingested or inhaled radioactivity. The next section describes how we apportion this risk estimate for whole-body exposure when considering the risks following the exposure of specific organs.

G.2.9 Organ Risks

For most sources of environmental contamination, inhalation and ingestion of radioactivity are more common than direct exposure. In many cases, depending on the chemical and physical characteristics of the radioactive material, inhalation and ingestion result in a nonuniform distribution of radioactive materials within the body so that some organ systems receive much higher doses than others. For example, iodine isotopes concentrate in the thyroid gland, and the dose to this organ can be orders of magnitude larger than the average dose to the body.

Fatal Cancer at Specific Sites

To determine the probability that fatal cancer occurs at a particular site, we have performed life table analyses for each cancer type using the information on cancer incidence and mortality in NAS80. For cancer other than leukemia and bone cancer, we used NAS80 Table V-14 (Age Weighted Cancer Incidence by Site Excluding Leukemia and Bone Cancer) and NAS80 Table V-15, which lists the BEIR Committee's estimates of the ratio of cancer fatality to cancer incidence for these various organs. The proportions of leukemia and fatal bone cancer caused by low-LET radiation were estimated using the results given in Tables V-17 and V-20 of NAS80. Normalized results, which give the proportion of fatal cancer caused by radiogenic cancer at a particular site, are listed in Table G-3. As noted above, these proportions are assumed to be the same for the BEIR-3 linear quadratic dose response model.

Information on the proportion of fatal cancers resulting from cancer at a particular organ is not precise. One reason is that the data in NAS80 (and Table G-3) are based on whole-body exposures, and it is possible that the incidence of radiogenic cancer varies depending on the number of exposed organs. Except for breast and thyroid cancer, very little information is available on radiogenic cancer resulting from exposure of only one region in the body. Another reason is that most epidemiology studies use mortality data from death certificates, which often provide questionable information on the site of the primary cancer. Moreover, when the existing data are subdivided into specific cancer sites, the number of cases becomes small, and sampling variability is increased. The net result of these factors is that numerical estimates of the total cancer risk are more reliable than those for most single sites.

The 1977 UNSCEAR Committee's estimated risks (UNSCEAR77) to different organs are shown in Table G-4. For all of the organs, except the breast, a high and low estimate was made. This range varies by a factor of 2 or more for most organs, Table G-4. Other site-specific estimates show a similar degree of uncertainty (Ka82), and it is clear that any system for allocating the risk of fatal cancer on an

TABLE G-3:
Proportion of the total risk of fatal radiogenic cancer resulting from cancer at a particular site

Site	Proportion of total risk[(a)]
Lung	0.21
Breast[(b)]	0.13
Red bone marrow[(c)]	0.16
Thyroid	0.099
Bone	0.009
Liver	0.085
Stomach	0.084
Intestines	0.039
Pancreas	0.058
Kidneys and urinary tract	0.025
Other[(d)]	0.11

[(a)] NAS80--Lifetime exposure and cancer expression; results are rounded to two figures.

[(b)] Average for both sexes.

[(c)] Leukemia.

[(d)] Total risk for all other organs, including the esophagus, lymphatic system, pharynx, larynx, salivary gland, and brain.

organ-specific basis is inexact. Table G-5 compares proportional risks by the NAS BEIR-3 Committee, UNSCEAR, and the ICRP. ICRP Report 26 provides organ-specific weights for assessing combined genetic and cancer risks from occupational exposure (ICRP77). In Table G-5, we have renormalized ICRP risks so that they pertain to cancer alone.

Considering that the cancer risk for a particular site is usually uncertain by a factor of 2 or more, as indicated by the range of UNSCEAR estimates in Table G-4, we would not expect perfect agreement in apportionment of total body risks. Table G-5, however, does indicate reasonable agreement among the three sets of estimates considered here.

TABLE G-4:
UNSCEAR estimates of cancer risks at specified sites

Site	Fatalities (per 10^6 person rad)	Average (per 10^6 organ rad)	Proportion of total risk
Lung	25-50	37.5	0.24
Breast[a]	25	25.0	0.16
Red bone marrow[b]	15-25	20.0	0.13
Thyroid	5-15	10.0	0.065
Bone	2-5	3.5	0.23
Liver	10-15	12.5	0.081
Stomach	10-15	12.5	0.081
Intestines	14-23	18.5	0.12
Pancreas	2-5	3.5	0.023
Kidneys and urinary tract	2-5	3.5	0.023
Other[c]	4-10	7.0	0.046

[a] Average for both sexes.
[b] Leukemia.
[c] Includes esophagus and lymphatic tissues.

Source: UNSCEAR77.

The differences between the proportions of the total risk of fatal cancer shown in Table G-5 are, for the most part, small in comparison to their uncertainty. We have used the BEIR-3 organ risks in preference to those made by other groups such as UNSCEAR or the ICRP for several reasons. BEIR estimates of organ risk are based on a projection of lifetime risk using age-specific risk coefficients, rather than just observations to date. Moreover, the 1980 BEIR Committee considered cancer incidence data as well as mortality data. This gives added confidence that the diagnostic basis for their estimates is correct. And, finally, because we apply these proportional organ risk estimates to the NAS80 cancer risk estimates for whole-body exposures, we believe it is consistent to use a single set of related risk estimates. The way we have used NAS80 to estimate mortality resulting from cancer at a particular site is outlined in the next section.

TABLE G-5:
Comparison of proportion of the total risk of radiogenic cancer fatalities by body organ

Site	NAS80[a]	UNSCEAR77	ICRP77[b]
Lung	.21	.24	.16
Breast	.13	.16	.20
Red Marrow	.16	.13	.16
Thyroid	.099	.065	.04
Bone	.009	.023	.04
Liver	.085	.081	(.08)[c]
Stomach	.084	.081	(.08)
Intestine	.039	.12	(.08)
Pancreas	.058	.023	(.08)
Kidneys and urinary tract	.025	.023	(.08)
Other	.11[d]	.046	---

[a] Lifetime exposure and cancer expression.
[b] Normalized for risk of fatal cancer (see text).
[c] Five additional organs which have the highest dose are assigned 0.08 for a total of 0.4.
[d] Others include esophagus, lymphatic system, pharynx, larynx, salivary gland, and brain.

G.2.10 Methodology for Calculating the Proportion of Mortality Resulting from Leukemia

Application of NAS80 to particular problems is straightforward but requires some familiarity with the details of that report. In this section we provide sample calculations based on the BEIR-3 linear dose response model for the case of fatal leukemia resulting from irradiation of the bone marrow throughout an average person's lifetime. We then compared this number to the average number of all fatal radiogenic cancers to obtain the proportion due to leukemia (Table G-3).

The NAS80 estimates in Table G-3 differ from the others in that they include both a consideration of age at exposure and a full expression of radiogenic cancer resulting from lifetime exposure. For example, Table V-17 in (NAS80) gives explicit age- and sex-dependent mortality coefficients for leukemia and bone cancer together.

The ratio of leukemia to bone cancer fatalities is given by the coefficient in the dose response relationship listed in Table V-17, i.e., 2.24/0.05. For lifetime exposure at a dose rate of one rad per year, Table V-17 lists 3,568 leukemia (and bone) deaths per 10^6 males and 2,709 deaths per 10^6 females (NAS80). Using a male-female birth ratio of 1.05 to 1.0, this averages to 3,149 fatal cancers per million persons in the general population. The total person rads causing these excess fatalities is the product of one rad per year, 10^6 persons, and 70.7 years (the average age of this population at death). Dividing the total number of fatalities by this product yields 44.5 fatalities per 10^6 person rad of which about 43.5 are due to leukemia. As noted above, for total body exposure, the average of the absolute and relative risk projection models yielded 280 premature cancer deaths per 10^6 person rad.

Therefore, P, the proportion of the whole-body risk caused by the lifetime risk of a leukemia death due to lifetime exposure of the red bone marrow, is:

$$P = \frac{43.5}{280} = .16 \quad \text{(cf. with Table G-3)} \qquad \text{(G-1)}$$

To obtain the proportional mortality for other cancers, we have used the site-specific, age-dependent risk coefficients in Table V-14 (NAS80) and the mortality ratios in Table V-15 to calculate the risk of fatal cancer from lifetime exposure at one rad per year (for each sex) and proceeded as in the example for leukemia outlined above.

To apply the data shown in Table G-3 to a particular organ, we multiply the average of the relative and absolute lifetime risk estimates for whole body lifetime exposure for a linear dose response, 280 fatalities per 10^6 person rad and 112 fatalities per 10^6 person rad for a linear quadratic response by the proportional mortality for that cancer. For example, using the linear model, a one rad dose (low-LET) to the kidney (urinary tract) resulting from lifetime exposure is estimated to cause a lifetime probability of death caused by radiogenic cancer that is equal to (.025) x (280×10^6) or 7×10^{-6}, i.e., 7 chances in a million.

Iodine-131 has been reported to be only 1/10 as effective as X-rays or gamma rays in inducing thyroid cancer (NAS72, NCRP77). For this cancer a linear dose response and a DREF of 10 is used in calculating lifetime probability of death. For example, the risk from a one rad dose to the thyroid from exposure to iodine-131 or iodine-129 is calculated as follows: (0.099) x (0.10) x (280×10^{-6}) or 2.8×10^{-6}, about 3 chances in a million.

G.2.11 Cancer Risks Due to Age-Dependent Doses

As noted in Appendix F, almost all of the dose models we have used are based on ICRP "Reference Man." ICRP dosimetric models are appropriate for adult workers and do not take into account differences resulting from the changes in physiological parameters between children and adults, e.g., intake rates, metabolism, and organ size. Although it is difficult to generalize for all radionuclides, in some cases these differences tend to counterbalance each other. For example, the ratio of minute volume to lung mass is relatively constant with age, i.e., within a factor of two, so that the ICRP adult model for insoluble materials provides a reasonably good estimate of the average annual dose throughout life.

An exception is the thyroid where the very young have a relatively high uptake of radioiodine into a gland which is much smaller than the adult thyroid, as noted in Table F-1. This results in a larger childhood dose and an increased risk which persists throughout life. Since this is a worst case situation, we have examined it with some care, using the age-specific risk coefficients for thyroid cancer in Table V-14 of the BEIR-3 report (NAS80) and the age-dependent dose model in ORNL84. For iodine-131 ingestion, the estimated lifetime risk is increased by a factor of 1.56 due to the 30 percent increase in lifetime dose over that obtained with the ORNL adult model, c.f. Appendix F. Results are about the same for inhalation of iodine-131--the estimated lifetime risk of fatal thyroid cancer is increased by a factor of 1.63 for ORNL's age-dependent dose estimate.

As noted in Appendix F, use of an age-dependent dosimetry for other radionuclides has yielded much smaller increased doses relative to adult models and therefore has little effect on estimates of lifetime risk. In particular, the lung dose and risk resulting from the inhalation of insoluble alpha particle emitters is nearly unchanged. The lifetime dose for an age-dependent dose model is only 1.09 times greater than that calculated using an adult model (Appendix F); the lifetime risk of lung cancer for this age-dependent model is a factor of 1.16 greater than we calculate for life exposure with the adult only model.

G.3 Fatal Cancer Risk Resulting from High-LET Radiations

In this section we explain how EPA estimates the risk of fatal cancer resulting from exposure to high-LET radiations. In some cases, ingestion and inhalation of alpha particle emitting radionuclides can result in a relatively uniform exposure of specific body organs by high-LET radiations. Unlike exposures to X-rays and gamma rays where the resultant charged particle flux results in linear energy transfers (LET) of the order of 0.2 to 2 keV per micron in tissue, 5 MeV alpha particles result in energy deposition at a track average rate of more than 100 keV per micron. High-LET radiations have a larger biological effect per unit dose (rad) than low-LET radiations. How much greater depends on the particular biological endpoint being considered. For cell killing and other readily observed endpoints, the relative biological effectiveness (RBE) of high-LET alpha radiations is often 10 or more times greater than low-LET radiations.

G.3.1 Quality Factors for Alpha Particles

Charged particles have been assigned quality factors, Q, to account for their efficiency in producing biological damage. Unlike an RBE value, which is for a specific and well-defined endpoint, a quality factor is based on an average overall assessment by radiation protection experts of potential harm of a given radiation relative to X or gamma radiation. In 1977, the ICRP assigned a quality factor of 20 to alpha particle irradiation from radionuclides (ICRP77). The reasonableness of this numerical factor for fatal radiogenic cancers at a particular site is not well known, but it is probably conservative for all sites and highly conservative for some.

The dose equivalent (in rem) is the dose, in rad, times the appropriate quality factor for a specified kind of radiation. For the case of internally deposited alpha particle emitters the dose equivalent from a one-rad dose is equal to 20 rem. It should be noted that prior to ICRP Report 26 (ICRP79), the quality factor for alpha particle irradiation was 10. That is, the biological effect from a given dose of alpha particle radiation was estimated to be 10 times that from an acute dose of low-LET X-rays or gamma rays of the same magnitude in rad. The ICRP decision to increase this quality factor to 20 followed from their decision to estimate the risk of low-LET radiations, in occupational situations, on the assumption that biological effects were reduced at low dose rates for low-LET radiation. There is general agreement that dose rate effects do not occur for high-LET (alpha) radiations. The new ICRP quality factor for alpha particles of 20 largely compensates for the fact that their low-LET risks are now based on an assumed dose rate reduction factor of 2.5. This DREF has been addressed in preparing EPA estimates of the risk per rad for alpha particle doses described below, in Section G.3.3.

In 1980 the ICRP published a task group report "Biological Effects of Inhaled Radionuclides" which compared the results of animal experiments on radiocarcinogenesis following the inhalation of alpha particle and beta particle emitters (ICRP80). The task group concluded that "the experimental animal data tend to support the decision by the ICRP to change the recommended quality factor from 10 to 20 for alpha radiation."

G.3.2 Dose Response Function

In the case of high-LET radiation, a linear dose response is commonly observed in both human and animal studies and the response is not reduced at low dose rates (NCRP80). Some data on human lung cancer indicate that the carcinogenic response per unit dose of alpha radiation is higher at low doses than higher ones (Ar81, Ho81, Wh83); in addition, some studies with animals show the same response pattern (Ch81, Ul82). We agree with the NAS BEIR-3 Committee that, "For high-LET radiation, such as from internally deposited alpha-emitting radionuclides, the linear hypothesis is less likely to lead to overestimates of the risk and may, in fact, lead to underestimates" (NAS80). However, at low doses, departures from linearity are small compared to the uncertainty in the human epidemiological data, and we believe a linear response provides an adequate model for evaluating risks in the general environment.

A possible exception to a linear response is provided by the data for bone sarcoma (but not sinus carcinoma) among U.S. dial painters who have ingested alpha-emitting radium-226 (NAS80). These data are consistent with a dose-squared response (Ro78). Consequently, the NAS BEIR-3 Committee estimated bone cancer risk on the basis of both linear and quadratic dose response functions. However, as pointed out in NAS80, the number of U.S. dial painters at risk who received less than 1000 rad was so small that the absence of excess bone cancer at low doses is not statistically significant. Therefore, the consistency of this data with a quadratic (or threshold) response is not remarkable and, perhaps, not relevant to evaluating risks at low doses. In contrast to the dial painter data, the incidence of bone cancer following radium-224 irradiation, observed in spondylitics by Mays and Spiess (Ma83, NAS80), in a larger sample at much lower doses, is consistent with a linear response. Therefore, for high-LET radiations EPA has used a linear response function to evaluate the risk of bone cancer.

Closely related to the choice of a dose response function is what effect the rate at which a dose of high-LET radiation is delivered has on its carcinogenic potential. This is a very active area of current research. There is good empirical evidence, from both human and animal studies, that repeated exposures to radium-224 alpha particles is 5 times more effective in inducing bone sarcomas than a single exposure which delivers the same dose (Ma83, NAS80). The 1980 NAS BEIR Committee took this into account in their estimates of bone cancer fatalities which EPA is using. We do not know to what extent, if any, a similar enhancement of carcinogenicity may occur for other cancers resulting from internally deposited alpha particle emitters. Nevertheless, we believe the ICRP quality factor of 20 is conservative, even at low dose rates.

G.3.3 Assumptions Made by EPA for Evaluating the Dose from Alpha Particle Emitters

We have evaluated the risk to specific body organs by applying the ICRP quality factor of 20 for alpha radiations to the risk estimates for low dose rate low-LET radiations as described above. For some organs this quality factor may be too conservative. Several authors have noted that estimates of leukemia based on a quality factor of 20 for bone marrow irradiation overpredicts the observed incidence of leukemia in persons receiving thorotrast (thorium oxides) (Mo79) and in the U.S. radium dial painters (Sp83). Nevertheless, in view of the paucity of applicable human data and the uncertainties discussed above, the ICRP quality factor provides a reasonable and prudent way of evaluating the risk due to alpha emitters deposited within body organs.

All of EPA risk estimates for high-LET radiations are based on a linear dose response function. For bone cancer and leukemia we use the absolute risk projection model described in the previous section. For other cancers we use the arithmetic average of relative and absolute risk projections.

Table G-6 indicates the Agency's estimates of the risk of fatal cancer due to a uniform organ dose in various organs from internally deposited alpha particles. These estimates are for lifetime doses at a constant dose rate. It was prepared by multiplying the average risk (based on the linear model for a uniformly distributed whole-body dose of low-LET radiation and a dose rate effectiveness factor of 2.5) by a quality factor of 20 and then apportioning this risk by organ, as indicated in Table G-6.

This procedure was not followed for bone cancer. As outlined above, the risk estimate for this cancer in the BEIR-3 report is based on data for high-LET (alpha) radiation and a direct estimation of the effect of the alpha radiation per high-LET rad. Some readers may note that the risk estimate in Table G-6, about 20 bone cancer fatalities per 10^6 person rad, is less than the 27 fatalities listed in Table A-27 of (NAS80) for alpha particles. This is because the analysis in Appendix A of NAS80, but not Chapter V of that report, assumes that in addition to a 2-year minimum induction period, 27 years are available for cancer expression. This is usually not the case for doses received beyond middle age. Hence, the estimated lifetime risk is smaller when it is based on a life table analysis that considers lifetime exposure in conjunction with death from all causes.

G.4 Uncertainties in Risk Estimates for Radiogenic Cancer

As pointed out in the introduction of this appendix, numerical estimates of risks due to radiation are neither extremely accurate nor precise. A numerical evaluation of radiogenic cancer risks depends both on epidemiological observations and a number of *ad hoc* assumptions which are largely external to the observed data set. These assumptions

TABLE G-6:
Estimated number of cancer fatalities from a lifetime exposure to internally deposited ALPHA particle emitters

Site	Proportional risk[a]	Fatalities per 10^6 person rad[b]
Lung	.21	460
Breast[c]	.13	290
Red marrow[d]	.16	350
Thyroid	.099	220
Bone[e]	.009	20
Liver	.085	190
Stomach	.084	190
Intestine	.039	90
Pancreas	.058	130
Kidneys and urinary tract	.025	55
Other-Sum (total)	.11	250

[a] Proportion of whole body risk from Table G-3.
[b] Rounded to two figures.
[c] Average for both sexes.
[d] Leukemia.
[e] Bone endosteum as defined in ICRP-30 (ICRP79).

include such factors as the expected duration of risk expression and variations in radiosensitivity as a function of age and demographic characteristics. A major assumption is the shape and slope of the dose effects response curve, particularly at low doses where there is little or no epidemological data. In 1971, the BEIR Committee based its estimates of cancer risk on the assumption that effects at low doses are directly proportional to those observed at high doses, the so called linear-nonthreshold hypothesis. As described above in Section G.2, the BEIR-3 Committee considered three dose response models and indicated a preference for the linear quadratic model. The risk coefficients the BEIR-3 Committee derived for their linear quadratic model, and to a lesser extent their linear model, are subject to considerable uncertainty primarily because of two factors: 1) systematic errors in the estimated doses of the individual A-bomb survivors, and 2) statistical uncertainty because of the small number of cancers observed at various dose levels.

G.4.1 Uncertainty of the Dose Response Models Due to Bias in the A-bomb Dosimetry

Although the BEIR-3 Committee's choice of a linear-quadratic response has gained considerable attention, it may not be generally appreciated that the BEIR-3 Committee's numerical evaluations of dose response functions for cancer due to low-LET radiation were based exclusively on the cancer mortality of the A-bomb survivors. Unfortunately, the dosimetry for A-bomb survivors, on which the BEIR-3 Committee relied, has since been shown to have large systematic errors which serve to undermine the analyses made by the Committee. As outlined below, the mathematical analyses made by the Committee were "constrained" to meet certain *a priori* assumptions. These assumptions have since been shown to be doubtful.

A careful state-of-the-art evaluation of the dose to A-bomb survivors was carried out by investigators from Oak Ridge National Laboratory in the early 1960's (Au67, Au77). The results of these studies resulted in a "T65" dose being assigned to the dose (kerma) in free air at the location of each survivor for both gamma rays and neutrons. A major conclusion of the ORNL study was that the mix of gamma ray and neutron radiations was quite different in the two cities where A-bombing occurred. These results indicated that at Hiroshima, the neutron dose was more important than the gamma dose when the greater biological efficiency of the high-LET radiations produced by neutrons was taken into account. Conversely, the neutron dose at Nagasaki was shown to be negligible compared to the gamma dose for that range of doses where there were a significant number of survivors. Therefore, the 1980 BEIR Committee evaluated the cancer risks to the survivors at Hiroshima on the assumption that the combined effects of gamma rays and particularly neutrons caused the observed cancer response.

Since the BEIR-3 report was published, it has become evident that the organ doses due to neutrons at Hiroshima were overestimated by about an order of magnitude, at distances where most of the irradiated persons survived bomb blast and yet received significant doses, 1000-1500 meters. In fact, the neutron doses at Hiroshima are quite comparable to those previously assigned, at similar distances, to Nagasaki survivors (Ke81a, Ke81b, RERF83, RERF84). Moreover, there is now grounds to believe the T65 estimates of gamma ray doses in both cities are also incorrect (RERF83, RERF84). While several factors need further evaluation, reduction of the gamma dose to individual survivors due to the local shielding provided by surrounding structures, is significant. The important point, however, is that the overestimate of the neutron dose to the Hiroshima survivors led to the BEIR-3 Committee attributing most of the risk to neutrons rather than gamma-rays. Hence, they underestimated the risk for low-LET radiations by, as yet, an unknown amount.

For their analysis of the A-bomb survivor data, the BEIR-3 Committee expanded the equations for low-LET radiations listed in Section G.2, Table G-1, to include a linear dose response function for neutrons:

1) $P(d,D) = c_1d + k_1D$ (G-2)

2) $P(d,D) = c_2d^2 + k_2D$ (G-3)

3) $P(d,D) = c_3d + c_4d^2 + k_3D$ (G-4)

where d is the gamma dose and D is that part of dose due to high-LET radiations from neutron interactions. Note that in equation G-4 the linear-quadratic (LQ) response, has two linear terms, one for neutrons and one for gamma radiation. In analyzing approximately linear data in terms of equation G-4, the decision as to how much of the observed linearity should be assigned to the neutron or the gamma component, i.e, k_3 and c_3, respectively, is crucial. As shown below, the BEIR-3 Committee attributed most of the observed radiogenic cancer to a linear response from neutron doses which did not occur.

The BEIR-3 Committee's general plan was to examine the dose response for leukemia and for solid cancer separately to find statistically valid estimates of the coefficients $c_1 \ldots c_4$ and $k_1 \ldots k_3$ by means of regression analyses. The regressions were made after the data were weighted in proportion to their statistical reliability; thus, Hiroshima results dominate the analysis. The T65 neutron and gamma doses to individual survivors are highly correlated since both are strongly decreasing functions of distance. This makes accurate determination of the coefficients in equation G-4 by means of a regression analysis extremely difficult. In addition, there is considerable sampling variation in the A-bomb survivor data due to small sample size which exacerbates the regression problem. Herbert gives a rigorous discussion of these problems for the case of the A-bomb survivors (He83). Because of these and other problems, agreement between the observed response for solid cancers and that predicted by any of the dose response functions examined by the BEIR-3 Committee is not impressive. For example, goodness of fit, based on Chi square, ranges from 0.20 for equation G-3 to 0.23 for equation G-4, to 0.30 for equation G-2 (Table V-11 in NAS80). For leukemia, the goodness of fit between the observed data and that predicted by the regression analysis is better, e.g., 0.49 for equations G-2 and G-3 (Table V8 in NAS80).

The Committee analyzed the A-bomb survivor data in two separate sets, i.e., first leukemia and then all cancer excluding leukemia (solid cancers). Their treatment of these two cases was not equivalent. Unlike the analysis of solid cancers, the Committee's analysis of leukemia considered the Nagasaki and Hiroshima data separately. Their approach

(p. 342 in NAS80) appears to be based on an unpublished paper by Charles Land and a published report by Ishimaru et al. on estimating the RBE of neutrons by comparing leukemia mortality in Hiroshima to that in Nagasaki (Is79). Unlike the case for solid cancers, (see below), the Committee's regression analysis of the leukemia mortality data did provide stable values for all of the coefficients in equation G-4, and therefore an RBE for neutrons as a function of dose, as well as the ratio of the linear to the dose-squared terms for leukemia induction due to gamma rays, (c_3/c_4).

Estimating the linear-quadratic response coefficients for solid cancers proved to be less straightforward. When the BEIR-3 analysis attempted to fit the A-bomb survivor data on solid cancers to a linear-quadratic dose response function, they found that the linear response coefficient, c_3 in equation G-4, varied from zero to 5.6 depending on the dose range considered. Moreover, their best estimate of the coefficient for the dose-squared term in equation G-4, i.e., c_4, was zero, i.e., the best fit yielded a linear response. Therefore, it was decided that the observations on solid cancers were "not strong enough to provide stable estimates of low dose, low-LET cancer risk when analyzed in this fashion," (NAS80, p. 186).

As outlined in the BEIR-3 Report, the Committee decided to use a constrained regression analysis, that is, substitute some of the parameters for equation G-4 found in their analysis of leukemia deaths to the regression analysis of the dose response for solid cancers. That is, both the neutron RBE at low dose (the ratio of the coefficient k_3 to c_3) and the ratio of c_3 to c_4, as estimated from the leukemia data, were assumed to apply to the induction of fatal solid cancers. Regression analyses that are constrained in this manner can yield much higher estimates of precision than is warranted by the data, as discussed by Land and Pierce (La83). They can also be very misleading. Herbert has discussed this point in detail as it applies to the BEIR-3 regression analysis (He83). The BEIR-3 Committee's substitution of the results of the leukemia regression for the data on solid cancers allowed them to make stable estimates of c_3, c_4, and k_3. These estimates became the basis for the "preferred" linear quadratic risk estimates for solid cancers presented in NAS80, i.e., the $\overline{\text{LQ-L}}$ model, page 187. (The response models for solid cancers that are based on the Committee's constrained regression analysis are designated with a bar in their 1980 report, e.g., $\overline{\text{LQ-L}}$ and $\overline{\text{L-L}}$.)

Given the information discussed above, it is possible to see, at least qualitatively, how the high bias in the estimated T65 neutron dose to the Japanese survivors affects the 1980 BEIR Committee's "preferred" LQ estimates of the risk coefficients for leukemia. The Committee's age-adjusted risk coefficients for leukemia are listed in Table V-8 (NAS80, page 184). For the linear-quadratic response, k_3, the neutron risk coefficient is 27.5. Tables A-11 (NAS80, page 341) and V-6 (NAS80, page 152) provide the estimates of neutron and gamma doses to the bone marrow of

Hiroshima survivors that were used by the Committee. Substituting these doses in their risk equations (Table V-8) indicates that about 70 percent of the leukemia deaths were ascribed to the neutron dose component then thought to be present at Hiroshima. As noted above, subsequent research indicates that the high-LET dose due to neutrons was actually much smaller.

It is not possible to accurately quantify what effect the Committee's use of these same coefficients had on their analysis of the dose response for solid cancers. Equation V-10 for solid cancers, p. 187 in NAS80, indicates about 60 percent of the solid tumor response was attributed to the T65 neutron dose; but this is a minimum estimate that ignores the effect of the assumed neutron doses on the value of k_3 and the ratio of c_3 to c_4.

The BEIR-3 Committee's LQ-L model assumes an RBE of 27.8 at low doses. In the Committee's L-L linear response model, the assumed RBE is 11.3. Therefore, this linear model is considerably less sensitive to the neutron dose component, assumed by the Committee, than their LQ-L model. For either model, most of the A-bomb survivors' radiogenic cancer was ascribed to the T65 neutron doses at Hiroshima.

There is no simple way of adjusting the 1980 BEIR risk estimates to account for the risk they attributed to neutrons. Adjustment of neutron doses alone is clearly inappropriate, since there is good reason to believe that T65 estimates of the dose due to gamma rays are also subject to considerable change. Moreover, not all of the individuals in a given T65 dose category will, necessarily, remain grouped together after new estimates of neutron and gamma doses are obtained. Both the numerator and denominator in the ratio of observed to expected cases are subject to change and indeed could change in opposite directions, a fact not considered in some preliminary (and premature) analyses (St81). Nevertheless, it is reasonable to conclude that bias in the estimated neutron doses at Hiroshima has led to considerable uncertainty in the BEIR-3 risk estimates and also to a systematic underestimation of the risk due to low-LET radiations. For this reason we believe that estimates based on the more conservative linear dose response should be given considerable weight *vis a vis* those made using the BEIR-3 linear quadratic models.

G.4.2 Sampling Variation

In addition to the systematic bias in the BEIR-3 risk estimates for low-LET radiation outlined above, the precision of the estimated linear and quadratic risk coefficients in the BEIR-3 report is poor due to statistical fluctuations due to sample size. Recently, Land and Pierce have reevaluated the precision of the BEIR-3 linear quadratic risk estimates to take into account, at least partially, the Committee's use of a constrained regression analysis (La83). This new analysis indicates that for the BEIR-3 LQ-L model for leukemia, the standard deviation of the linear term is nearly as large as the risk coefficient itself (±0.9 compared to a risk coefficient of 1). For the LQ-L model, solid cancer, the standard deviation is ±1.5 compared to a risk coefficient of 1.6.

It is likely that at least part of the uncertainty attributed to sampling variation in the BEIR-3 risk estimates is not due to sample size and other random factors but rather due to the use of incorrect dose estimate for the A-bomb survivors. The correlation of neutron and gamma-ray doses has been a major underlying cause of the uncertainty in regression analysis using the T-65 doses. Analyses of revised data with much smaller neutron doses may result in better precision. At present, we have concluded that the BEIR-3 risk coefficients are uncertain by at least a factor of 2, see below, as well as being biased low by an additional factor of 2 or more.

G.4.3 Uncertainties Arising from Model Selection

In addition to a dose response model, a "transportation model" is needed to apply the risks from an observed irradiated group to another population having different demographic characteristics. A typical example is the application of the Japanese data for A-bomb survivors to western people. Seymore Jablon, (Director of the Medical Follow-up Agency of the National Research Council, NAS) has called this the "transportation problem," a helpful designation because it is often confused with the risk projection problem described below. However, there is more than a geographic aspect to demographic characteristics. The "transportation problem" includes estimating the risks for one sex based on data for another and a consideration of habits influencing health status such as differences between smokers and nonsmokers.

The BEIR-3 Committee addressed this problem in their 1980 report and concluded, based largely on the breast cancer evidence, that the appropriate way to transport the Japanese risk to the U.S. population was to assume that the absolute risk over a given observation period was transferable but that relative risk was not. Therefore, the Committee calculated what the relative risk would be if the same number of excess cancer deaths were observed in a U.S. population having the same age characteristics as the A-bomb survivors. The base line cancer rates in the U.S. and Japan are quite different for some specific cancers so this is a reasonable approach. However, it contains the assumption that while the cancer initiation process is the same in the two countries, the actual number of radiogenic cancers that actually occur is the result of cancer promotion, the latter being a culturally dependent variable.

An alternative approach to solving the "transportation problem" is that of the 1972 NAS BEIR-1 Committee. This Committee assumed relative risks would be the same in the United States and Japan and transferred the observed percentage increase directly to the U.S. population. We have compared estimates of the lifetime risk for these two treatments of the "transportation problem" in order to find out how sensitive the BEIR-3 Committee risk estimates are to their assumptions. To do this, we calculated new relative risk estimates for solid cancers based on the age-specific cancer mortality of the Japanese population rather than the U.S. data used by the BEIR-3 Committee. We found that this alternative approach did not have much effect on the estimated lifetime risk of solid radiogenic cancer, i.e., a change of 3 percent for males, and 17 percent for females. We have concluded that the amount of uncertainty introduced by transporting cancer risks observed in Japan to the U.S. population is small compared to other sources of uncertainty in this risk assessment. Base-line leukemia rates are about the same in the countries, so we believe these risks are also "transportable."

The last of the models needed to estimate risk is a risk projection model. As outlined in Section G.2, such models are used to project what future risks will be as an exposed population ages. For leukemia and bone cancer, where the expression time is not for a full lifetime but rather 25 years, absolute and relative risk projection models yield the same number of radiogenic cancers, but would distribute them somewhat differently by age. For solid cancers, other than bone, the BEIR-3 Committee assumed that radiogenic cancers would occur throughout the lifetime. This makes the choice of projection model more critical, because the relative risk projection yields estimated risks about three times larger than that obtained with an absolute risk projection, as shown in Table G-2. Because we have used the average of these two projections for solid cancers, we believe this reduces the uncertainty from the choice of model to about a factor of 2 or perhaps less, depending on the age distribution of fatal radiogenic cancer, as outlined in Section G.2 above.

Similarly, there is as yet insufficient information on radiosensitivity as a function of the age at exposure. The age-dependent risk coefficients we have used are those presented in the BEIR-3 report. As yet, there is little information on the ultimate effects of exposure during childhood. As the A-bomb survivors' population ages, more information will become available on the cancer mortality of persons irradiated when they were young. Table G-2 indicates that the more conservative BEIR-1 estimates for the effect of childhood exposures would increase BEIR-3 risk estimates by about 40 percent. As this is probably an upper limit, the lack of more precise information is not a major source of uncertainty in estimates of the risk caused by lifetime exposure. Similarly, the BEIR-3 Committee did not calculate population risks for radiogenic cancer that included in utero radiation because they felt the available data were unreliable. We have deferred to their judgment in this regard. The BEIR-1 report did include in utero cancer risk. These had little effect, 1 to 10 percent, on the lifetime risk of cancer from lifetime exposure. An effect this small is not significant relative to other sources of uncertainty in the risk assessment.

Summary

We can only semi-quantitatively estimate the overall uncertainty in the risk per rad for low-LET radiations. We expect that more quantitative estimates of the uncertainty will be possible only after the A-bomb dose reassessment is completed and the A-bomb survivor data reanalyzed on the basis of the new dose estimates. It should be noted, however, that even if all systematic bias is removed from the new dose estimates, there will still be considerable random error in the dose estimate for each survivor. This random error biases the estimated slope of the dose response curve so that it is smaller than the true dose response (Da72, Ma59). The amount of bias introduced depends on the size of the random error in the dose estimates and their distribution which are unknown quantities at this stage of the dose reassessment.

The source of uncertainty in risk estimates for low-LET radiations can be ranked as shown in Table G-7.

TABLE G-7:
A ranking of causes of uncertainty in estimates of the risk of cancer

Source of uncertainty	Degree of uncertainty
Choice of dose response model	±250 percent(a)
Slope of dose response resulting from sampling variation	±200 percent(b)
Choice of an average risk projection model	±100 percent(c)
Choice of transportation model	±20 percent(d)
A-bomb T-65 dosimetry	Plus only, amount unknown

(a) For choices limited to BEIR-3 linear and linear quadratic models, see G.2.
(b) Estimate of 2 standard deviations for the BEIR-3 $\overline{LQ}$ model (La83).
(c) Average of relative and absolute projection as described above.
(d) For the total of all cancers, not specific cancers.

The estimates of uncertainty in Table G-7 are not wholly comparable and must be interpreted carefully. However, they do have some illustrative value, particularly when ordered in this way. The uncertainty listed for the slope of dose response is a nominal value for the BEIR-3 linear quadratic $\overline{LQ}$ formulation (La83) in that it is only valid insofar as the Committee's assumptions are true. It is based on a two standard deviation error so that the expectation that the error is less than indicated is 95 percent. We do not believe the uncertainty in the BEIR-3 linear estimate, $\overline{L\text{-}L}$, is significantly smaller, c.f. Tables V-9 and V-11 in NAS80.

The other uncertainties listed in Table G-7 are quite different, being more in the nature of informed judgments than the result of a statistical analysis. It is doubtful that all radiogenic cancers have the same type of response functions. However, if they were all linear, as breast cancer and thyroid appear to be, the BEIR-3 linear quadratic response model would underestimate the response by 250 percent. If most cancers have a linear quadratic response, or equivalently, a dose rate reduction factor equal to the difference in slope at low doses between the BEIR-3 linear and linear quadratic models, the use of a linear model would overestimate the response by a factor of 2.5. We believe that a factor of 250 percent is a conservative estimate of the uncertainty introduced by the lack of data at low dose rates.

As discussed above, the uncertainty due to the choice of an absolute or a relative risk model is about a factor of 3. The use of the average risk for these two models reduces the uncertainty in risk projection by more than a factor of 2, since it is known that a relative risk projection is high for some kinds of cancer and that an absolute risk projection is low for others.

The uncertainties listed in Table G-7 are largely independent of each other and therefore unlikely to be correlated in sign. Their root mean square sum is about 300 percent, indicating the expectation that calculated risks would be within a factor of 3 or so of the true value. This result is overly optimistic because it does not include consideration of the uncertainty introduced by the bias in the A-bomb dosimetry or by the constrained regression analysis used by the BEIR-3 Committee.

G.5 Other Radiation-Induced Health Effects

The earliest report of radiation-induced health effects was in 1896 (Mo67), and it dealt with acute effects in skin caused by x-ray exposures. Within the six-year period following, 170 radiation-related skin damage cases had been reported. Such injury, like many other acute effects, is the result of exposure to hundreds or thousands of rad. Under normal environmental exposure situations, however, such exposure conditions are not possible and therefore will not be considered in assessing the risk to the general population from radionuclide releases.

Although radiation-induced carcinogenesis was the first delayed health effect reported, radiation-induced genetic changes were reported early too. In 1927, H.J. Muller reported on x-ray induced mutations in animals and in 1928, L.J. Stadler reported a similar finding in plants (Ki62). At about the same time, radiation effects on the developing embryo were reported. Case reports in 1929 showed a high rate of microcephaly (small head size) and central nervous system disturbance and one case of skeletal defects in children irradiated in utero (UNSCEAR69). These effects, at unrecorded but high exposures, appeared to produce central nervous system and eye defects similar to those reported in rats as early as 1922 (Ru50).

For purposes of assessing the risks of environmental exposure from radionuclide releases, the genetic effects and in utero developmental effects are the only health hazards other than cancer that are addressed in this appendix.

G.5.1 Types of Genetic Harm and Duration of Expression

Genetic harm or the genetic effects of radiation exposure are those effects induced in the germ cells (eggs or sperm) of exposed individuals, which are transmitted to and expressed only in their progeny and future generations.

Of the possible consequences of radiation exposure, the genetic risk is more subtle than the somatic risk. Genetic risk is incurred by fertile people when radiation damages the nucleus of the cells which become their eggs or sperm. The damage, in the form of a mutation or a chromosome aberration, is transmitted to, and may be expressed in, a child conceived after the radiation exposure and in subsequent generations. However, the damage may be expressed only after many generations, may be lost by chance, or, alternately, it may never be expressed because of failure to reproduce.

EPA treats genetic risk as independent of somatic risk because, although somatic risk is expressed in the person exposed, genetic risk is expressed only in progeny and, in general, over many subsequent generations. Moreover, the types of damage incurred often differ in kind from cancer and cancer death. Historically, research on genetic effects and development of risk estimates has proceeded independently of the research on carcinogenesis. Neither the dose response models nor the risk estimates of genetic harm are derived from data on studies of carcinogenesis.

Although genetic effects may vary greatly in severity, the genetic risks considered by the Agency evaluating the hazard of radiation exposure include only those "disorders and traits that cause a serious handicap at some time during lifetime" (NAS80). Genetic risk may result from one of several types of damage that ionizing radiation can cause in the DNA within eggs and sperm. The types of damage usually considered are: dominant and recessive mutations in autosomal chromosomes, mutations in sex-linked (x-linked) chromosomes, chromosome aberrations (physical rearrangement or removal of part of the genetic message on the chromosome or abnormal numbers of chromosomes), and irregularly inherited disorders (genetic conditions with complex causes, constitutional and degenerative diseases, etc.).

Estimates of the genetic risk per generation are based on a 30-year reproductive generation. That is, the median parental age for production of children is age 30 (one-half the children are produced by persons less than age 30, the other half by persons over age 30). Thus, the radiation dose accumulated up to age 30 is used to estimate the genetic risks. Using this accumulated dose and the number of live births in the population along with the estimated genetic risk per unit dose, it is possible to estimate the total number of genetic effects per year, those in the first generation and the total across all time. Most genetic risk analyses have provided such data. EPA assessment of risks of genetic effects includes both first generation estimates and total genetic burden estimates.

Direct and Indirect Methods of Obtaining Risk Coefficients for Genetic Effects

Genetic effects, as noted above, may occur in the offspring of the exposed individuals or they may be spread across all succeeding generations. Two methods have been used to estimate the frequency of mutations in the offspring of exposed persons, direct and indirect. In either case, the starting point is data from animal studies, not data obtained from studies of human populations.

For a direct estimate, the starting point is the frequency of a mutation per unit exposure in some experimental animal study. The 1982 UNSCEAR (UNSCEAR82) report gave an example of the direct method for estimating induction of balanced reciprocal translocations (a type of chromosomal aberration) in males per rad of low level, low-LET radiation.

			Induction rate/rad
1)	Rate of induction in rhesus monkey Spermatogonia: cytogenetic data		0.86×10^{-4}
2)	Rate of induction that relates to recoverable translocations in the F_1 (1st filial generation) progeny [divide (1) by 4]		0.215×10^{-4}
3)	Rate after low dose rate X-rays: based on mouse cytogenetic observations [divide (2) by 2]		0.1075×10^{-4}
4)	Rate after chronic gamma-irradiation: based on mouse cytogenetic observations [divide (2) by 10]		0.022×10^{-4}
5)	Expected rate of unbalanced products: [multiply (3) and (4) by 2]	for (3)	0.215×10^{-4}
		for (4)	0.043×10^{-4}
6)	Expected frequency of congenitally malformed children in the F_1, assuming that about 6 percent of unbalanced products [item (5) above] contribute to this		
		for low dose rate X-rays	1.3×10^{-6}
		for chronic gamma radiation	$\sim 0.3 \times 10^{-6}$

For humans, UNSCEAR estimates that as a consequence of induction of balanced reciprocal translocations in exposed fathers, an estimated 0.3 to 1.3 congenitally malformed children would occur in each 10^6 live births for every rad of parental radiation exposure.

A complete direct estimate of genetic effects would include estimates, derived in a manner similar to that shown above for each type of genetic damage. These direct estimates can be used to calculate the risk of genetic effects in the first generation (F_1) children of exposed parents.

The indirect (or doubling dose) method of estimating genetic risk also uses animal data but in a different way. The 1980 BEIR-3 report (NAS80) demonstrates how such estimates are obtained.

1)	Average radiation induced-mutation per gene for both sexes in mice [based on 12 locus data in male mice]: induction rate per rad	0.25×10^{-7}
2)	Estimated human spontaneous mutation rate per gene	0.5×10^{-6} to 0.5×10^{-5}
3)	Relative mutation risk in humans [divide (1) by (2)]	0.005 to 0.05
4)	Doubling dose: the exposure needed to double the human mutation rate	200 to 20 rad

The doubling dose can then be used to estimate the equilibrium genetic effects or the genetic burden in all future generations caused by the exposure of parents. Since the genetic component of congenital defects occurring in the population can be estimated by epidemiological surveys, and this component is considered to be maintained at an equilibrium level by mutations, a doubling dose of ionizing radiation would double these genetic effects. Dividing the number of the various genetic effects in 10^6 live-births by the doubling dose yields the estimate of genetic effects per rad. For example:

1)	Autosomal dominant and x-linked diseases, current incidence	10,000 per 10^6 live births
2)	Estimated doubling dose	20 to 200 rad
3)	Estimate of induced autosomal dominant and x-linked diseases	50 to 500 per 10^6 live births per rad of parental exposure.

A doubling dose estimate assumes that the total population of both sexes is equally irradiated, as occurs from background radiation, and that the population exposed is large enough so that all genetic damage can be expressed in future offspring. Although it is basically an estimate of the total genetic burden across all future generations, it can also provide an estimate of effects that occur in the first generation. Usually a fraction of the total genetic burden for each type of damage is assigned to the first generation using population genetics data as a basis to determine the fraction. For example, the BEIR-3 committee geneticists estimated that one-sixth of the total genetic burden of x-linked mutations would be expressed in the first generation, five-sixths across all future generations. EPA assessment of risks of genetic effects includes both first generation estimates and total genetic burden estimates.

G.5.2 Estimates of Genetic Harm Resulting from Low-LET Radiations

One of the first estimates of genetic risk was made in 1956 by the NAS Committee on the Biological Effects of Atomic Radiation (BEAR Committee). Based on *Drosophila* (fruit fly) data and other considerations, the BEAR Genetics Committee estimated that 10 Roentgens (10 R*) per generation continued indefinitely would lead to about 5,000 new instances of "tangible inherited defects" per 10^6 births, and about one-tenth of them would occur in the first generation after the irradiation began (NAS72). The UNSCEAR addressed genetic risk in their 1958, 1962, and 1966 reports (UNSCEAR58, 62, 66). During this period, they estimated one rad of low-LET radiation would cause a 1 percent to 10 percent increase in the spontaneous incidence of genetic effects.

In 1972, both the NAS BEIR Committee (NAS72) and UNSCEAR (UNSCEAR72) reexamined the question of genetic risks. Although there were no definitive human data, additional information was available on the genetic effects of radiation on mammals and insects. In 1977, UNSCEAR reevaluated the 1972 genetics estimates (UNSCEAR77). Their new estimates used recent information on the current incidence of various genetic conditions, along with additional data on radiation exposure of mice and marmosets and other considerations.

In 1980, an ICRP Task Group (ICRPTG) summarized recommendations that formed the basis for the genetic risk estimates published in ICRP Report 26 (Of80). These risk estimates are based on data similar to that used by the BEIR and UNSCEAR Committees, but with slightly different assumptions and effect categories, Table G-8.

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*R is the symbol for Roentgen, a unit of measurement of x-radiation, equivalent to an absorbed dose in tissue of approximately 0.9 rad.

Table G-8. ICRP task group estimate of number of cases of serious genetic ill health in liveborn from parents irradiated with 10^6 man-rem in a population of constant size[a] (Assumed doubling dose = 100 rad)

Category of genetic effect	First generation	Equilibrium
Unbalanced translocations: risk of malformed liveborn	23	30
Trisomics and XO	30	30
Simple dominants and sex-linked mutations	20	100
Dominants of incomplete penetrance and multifactorial disease maintained by mutation	16	160
Multifactorial disease not maintained by mutation	0	0
Recessive disease	--	--
Total	89	320

[a] This is equivalent to effects per 10^6 liveborn following an average parental population exposure of 1 rem per 30-year generation, as used by BEIR and UNSCEAR.

Source: Of80.

The 1980 NAS BEIR Committee revised genetic risk estimates BEIR-3 (NAS80). The revision considered much of the same material that was in BEIR-1 (NAS72), the newer material considered by UNSCEAR in 1977 (UNSCEAR77), and some additional data. Estimates for the first generation are about a factor of 2 smaller than reported in the BEIR-1 report. For all generations, the new estimates are essentially the same, Table G-9.

The most recent genetic risk estimate, in the 1982 UNSCEAR Report (UN82), includes some new data on cells in culture and the results of genetic experiments using primates rather than rodents, Table G-10.

Although all of the reports described above used somewhat different sources of information, there is reasonable agreement in the estimates (Table G-11). Most of the difference is caused by the newer information used in each report. Note that all estimates listed above are based on the extrapolation of animal data to humans. Groups differ in their interpretation of how genetic experiments in animals might be expressed in humans. While there are no comparable human data at present, information on hereditary defects among the children of A-bomb survivors provides a degree of confidence that the animal data do not lead to underestimates of the genetic risk following exposure to humans. (See "Observations on Human Populations" which follows.)

Table G-9. BEIR-3 estimates of genetic effects of an average population exposure of 1 rem per 30-year generation

Type of genetic disorder	Current incidence per 10^6 liveborn	Effects per 10^6 liveborn per rem per generation	
		First Generation	Equilibrium
Autosomal dominant and x-linked	10,000	5-65	40-200
Irregularly inherited	90,000	(not estimated)	20-900
Recessive	1,100	Very few	Very slow increase
Chromosomal aberrations	6,000	Fewer than 10	Increases only slightly
Total	107,100	5-75	60-1100

Source: NAS80.

Table G-10. UNSCEAR 1982 estimated effect of 1 rad per generation of low dose or low dose rate, low-LET radiation on a population of 10^6 liveborn according to the doubling dose method (Assumed doubling dose = 100 rad)

Disease classification	Current incidence	Effect of 1 rad per generation	
		First Generation	Equilibrium
Autosomal dominant and x-linked diseases	10,000	15	100
Recessive diseases	2,500	Slight	slow increase
Chromosomal diseases			
Structural	400	2.4	4
Numerical	3,000	Probably very small	
Congenital anomalies, anomalies expressed later, constitutional and degenerative diseases	90,000	4.5	45
Total	105,900	22	149

Source: (UNSCEAR82).

Table G-11. Summary of genetic risk estimates per 10^6 liveborn for an average population exposure of 1 rad of low dose or low dose rate, low-LET radiation in a 30-year generation

Source	Serious hereditary effects First generation	 Equilibrium (all generations)
BEAR, 1956 (NAS72)	---	500
BEIR-I, 1972 (NAS72)	49[a] (12-200)	300[a] (60-1500)
UNSCEAR, 1972 (UNSCEAR72)	9[a] (6-15)	300
UNSCEAR, 1977 (UNSCEAR77)	63	185
ICRPTG, 1980 (Of80)	89	320
BEIR-3, 1980 (NAS80)	19[a] (5-75)	257[a] (60-1100)
UNSCEAR, 1982 (UNSCEAR82)	22	149

Numbers in parentheses are the range of estimates.

[a] Geometric Mean is calculated by taking the square root of the product of two numbers for which the mean is to be calculated. The cube root of three numbers, etc. In general, it is the N^{th} root of the product of N numbers for which the mean is to be calculated.

It should be noted that the genetic risk estimates summarized in Table G-11 are for low-LET, low dose, and low dose rate irradiation. Much of the data were obtained from high dose rate studies, and most authors have used a sex-averaged factor of 0.3 to correct for the change from high dose rate, low-LET to low dose rate, low-LET exposure (NAS72, 80, UNSCEAR72, 77). However, factors of 0.5 to 0.1 have also been used in estimates of specific types of genetic damage (UNSCEAR72,77,82).

G.5.3 Estimates of Genetic Harm for High-LET Radiations

Although genetic risk estimates are made for low-LET radiation, some radioactive elements, deposited in the ovary or testis can irradiate the germ cells with alpha particles. The ratio of the dose (rad) of low-LET radiation to the dose of high-LET radiation producing the same endpoint is called RBE and is a measure of the effectiveness of high-LET compared to low-LET radiation in causing the same specific endpoint.

Studies with the beta particle emitting isotopes carbon-14 and tritium yielded RBEs of 1.0 and 0.7 to about 2.0, respectively (UNSCEAR82). At the present time, the RBE for genetic endpoints due to beta particles is taken as one (UNSCEAR77,82).

Studies of the RBE for alpha-emitting elements in germinal tissue have used only plutonium-239. Studies comparing cytogenetic endpoints after chronic low dose rate gammma radiation exposure, or incorporation of plutonium-239 in the mouse testis, have yielded RBEs of 23 to 50 for the type of genetic injury (reciprocal translocations) that might be transmitted to liveborn offspring (NAS80, UNSCEAR77,82). However, an RBE of 4 for plutonium-239 compared to chronic low-LET radiation was reported for specific locus mutations observed in neonate mice (NAS80). Neutron RBE, determined from cytogenetic studies in mice, also ranges from about 4 to 50 (UNSCEAR82, Gr83a, Ga82). Most reports use an RBE of 20 to convert risk estimates for low dose rate, low-LET radiation to risk estimates for high-LET radiation.

G.5.4 Uncertainty in Estimates of Radiogenetic Harm

Chromosomal damage and mutations have been demonstrated in cells in culture, in plants, in insects, and in mammals (UNSCEAR72,77,82). Chromosome studies in peripheral blood lymphocytes of persons exposed to radiation have shown a dose-related increase in chromosome aberrations (structural damage to chromosome) (UNSCEAR82). In a study of nuclear dockyard workers exposed to external x-radiation at rates of less than 5 rad per year, Evans, et al. (Ev79) found a significant increase in the incidence of chromosome aberrations. The increase appeared to have a linear dependence on cumulative dose. In a study of people working and living in a high natural background area where there was both external gamma-radiation and internal alpha-radiation, Pohl-Ruling et al. (Po78) reported a complex dose response curve. For mainly gamma-radiation exposure (less than 10 percent alpha radiation), they reported that the increase in chromosome aberrations increased linearly from 100 to 200 mrad per year then plateaued from 300 mrad to 2 rad per year. They concluded:

> "From these data, and data in the literature, it can be concluded that the initial part of the dose-effect curve for chromosome aberrations is not linear or sigmoid with a threshold at the lowest dose, but rises sharply and passes into a complex upward form with a kind of plateau until it meets the linear curve of the high dose."

Although chromosomal damage in peripheral blood lymphocytes cannot be used for predicting genetic risk in progeny of exposed persons, it is believed by some to be a direct expression of the damage, analogous to that induced in germ cells, resulting from the radiation exposure. It is at least evidence that chromosome damage can occur *in vivo* in humans.

Although evidence from the genetic studies in Japan falls short of providing statistically significant conclusions, all indicators support the hypothesis that genetic damage resulted from the radiation exposure (Sc81b, Sa82). While these data give rise to estimates of the doubling dose (see below) and probabilities of mutation per locus per rad that have large errors, there is no reason to believe that they are biased, and they are adequate to recommend the elimination of some previously conjectured values. However, in the sources of numerical risk estimates used in this document, human genetic risks following radiation exposure are based on extrapolations from animal data. As genetic studies proceeded, emphasis has shifted from *Drosophila* (fruit flies) to mammalian species in attempts to find an experimental system which would reasonably project what might happen in humans.

For example, Van Buul (Va80) reported the slope (b) of the linear regression, $Y = a + bD$, for induction of reciprocal translocations in spermatogonia (one of the stages of sperm development) in various species as follows:

	$b \times 10^4 \pm sd \times 10^4$
Rhesus monkey	0.86 ± 0.04
Mouse	1.29 ± 0.02 to 2.90 ± 0.34
Rabbit	1.48 ± 0.13
Guinea Pig	0.91 ± 0.10
Marmoset	7.44 ± 0.95
Human	3.40 ± 0.72

These data indicate that animal-based estimates for this type of genetic effect would be within a factor of 4 of the true human value. In this case most of the animal results would underestimate the risk in humans.

However, when risk estimates such as this are used in direct estimation of risk for the first generation, the total uncertainty in the estimate becomes indeterminate. Even if studies have been made in a species which can predict the dose response and risk coefficient for a specific-radiation induced genetic damage, there is no certainty that it predicts the response for all genetic damage of that type. In addition, as shown in the example from the 1982 UNSCEAR report (UNSCEAR82) in Section G.5.1, additional assumptions based on observations, usually in other animal species, are used to adjust the risk coefficient to what is expected for humans. The uncertainty in these extrapolations has not been quantified.

A rough estimate of the uncertainty can be obtained by comparing direct estimates of risk for the first generation with doubling-dose estimates in the 1977 UNSCEAR report (UNSCEAR77). The estimates differ by a factor between 2 and 6 with the direct estimate usually smaller than the doubling dose estimate.

A basic assumption in the doubling-dose method of estimation is that there is a proportionality between radiation induced and spontaneous mutation rates. Some of the uncertainty was removed in the 1982 UNSCEAR report with the observation that in two test systems (fruit flies and bacteria), there is a proportionality between spontaneous and induced mutation rates at a number of individual gene sites. There is still some question as to whether the sites that have been examined are representative of all sites and all gene loci or not. The doubling dose estimate dose, however, seems better supported than the direct estimate.

While there is still some uncertainty as to what should be doubled, future studies on genetic conditions and diseases can only increase the total number of such conditions. Every report, from the 1972 BEIR and 1958 UNSCEAR reports to the most recent, has listed an increased number of conditions and diseases which have a genetic component.

Observations on Human Populations

As noted earlier, the genetic risk estimates are based on interpretation of animal experiments as applied to data on naturally-occurring hereditary diseases and defects in man. A study of birth cohorts was initiated in the Japanese A-bomb survivors in mid-1946. This resulted in a detailed monograph by Neel and Schull (Ne56) which outlined the background of the first study and made a detailed analysis of the findings to January 1954 when the study terminated. The authors concluded ". . . under circumstances where, on the basis of what is known concerning the radiation genetics of mammals, it appears unlikely that conspicuous genetic effects of the atomic bombs could be demonstrated, such effects have in fact not been demonstrated. The present study can in no way be interpreted to mean that there were no mutations induced in the survivors of the atomic blasts. Neither, on the other hand, is the reverse interpretation - that of mutation production - permissible from this series of observations . . .. We are left with inconclusive findings, albeit findings which permit us to set confidence limits." (Ne56).

Further, it appeared that it was improbable that human genes were so sensitive that exposures as low as 3 R, or even 10 R, would double the mutation rate. While this first study addressed a number of endpoints such as : sex ratio, malformations, pre- and perinatal mortality and anthropometric data, subsequent studies have addressed these and other endpoints. The most recent reports on this birth cohort of 70,082 persons have attempted to estimate the minimum doubling dose for genetic effects in humans (Sc81a, Sa82).

Data on four endpoints have been reviewed for this birth cohort. Frequency of stillbirths, major congenital defects, prenatal death, and frequency of death prior to age 17 have been examined in the entire cohort. Frequency of cytogenetic aberrations (sex chromosome aneuploidy) and frequency of biochemical variants (a variant enzyme or protein electrophoresis pattern) have been measured on large subsets of this cohort.

Although the updated data reported appear to suggest radiation effects have occurred, the numbers are small and not statistically significant. Overall, the estimated doubling dose for low-LET radiation at high doses and dose rates for human genetic effects is about 156 rem (Sc81a) or 250 rem (Sa82). As noted above, animal studies indicate that chronic exposures to low-LET radiation would be less hazardous by a factor of 3 (NAS72, 80). This would increase the estimated doubling dose to 468 rem to 750 rem, respectively. These recent reports suggest the minimum doubling dose for humans may be 4 to 7 times higher than those in Table G-11 (based on animal data). It would be premature to reach a firm decision on the exact amount since these reports are based on the T65D dosimetry in Japan (see Section G.2) which is being revised. However, we believe EPA estimates of genetic risks will prove to be very conservative even when the dosimetry of A-bomb survivors is revised.

EPA is using the geometric mean of the BEIR-3 range of doubling doses, about 110 rad. The minimum doubling dose reported above is 4 to 7 times greater. It is unlikely that dose estimates for Japanese survivors will change by this much (RERF83, 84). Therefore, EPA believes the estimate of a doubling of about 100 rad will continue to be a conservative estimate.

Ranges of Estimates Provided by Various Models

EPA has continued to follow the recommendations of the 1980 BEIR-3 committee and use a linear nonthreshold model for estimating genetic effects. Although, as pointed out by the 1982 UNSCEAR committee, there are a number of models other than linear ($Y = c + ad$), e.g., linear quadratic ($Y = c + bD + eD^2$), quadratic ($Y = k + fD^2$), even power function ($Y = k + gD^h$)*. However, there are strong data to support the hypothesis that mutations themselves are single track events. That is, the mutations follow a linear dose response function while the observed mutation rate shows the influence of other factors, and may be nonlinear (UNSCEAR82).

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*Y is yield of genetic effects; D is radiation dose; c, C, k, and K are spontaneous incidence constants for genetic effects; and a, b, e, f, g, and h are the rate constants for radiation induced genetic effects.

Most of the arguments for a nonlinear dose response have been based on target theory (Le62) or microdosimetric site theory (Ke72). However, other theories based on biology [e.g., enzyme induction-saturation (Go80,82), repair-misrepair (To80)] could also provide models that fit the observed data. There is still much disagreement on which dose response model is appropriate for estimating genetic effects in humans. Until there is more consensus, the linear nonthreshold model appears to be a prudent approach that will not underestimate the risks.

The agreement in estimates made on a linear nonthreshold model in various reports is reasonably good. Even though the authors of the reports used different animal models, interpreted them in different ways, and had different estimates of the level of human genetic conditions in the population, the range of risk coefficients is about an order of magnitude (see Table G-11). For the most recent, more comparable estimates, the range is a factor of 2 to 4 (see ICRPTG, BEIR-3 and UNSCEAR 1982 in Table G-11).

G.5.5 The EPA Genetic Risk Estimate

There is no compelling evidence for preferring any one set of the genetic risk estimates listed in Table G-11. EPA has used the estimates from BEIR-3 (NAS80). These "indirect" estimates are calculated using the normal prevalence of genetic defects and the dose that is considered to double this risk. The NAS estimates which EPA uses are based on a "doubling dose" range with a lower bound of 50 rem and an upper bound of 250 rem. We prefer these risk estimates to those made by the ICRP task group (Of80), which used a "direct" estimate because the ICRPTG tabulation combines "direct" estimates for some types of genetic damage with doubling dose estimates for others. We also prefer the BEIR-3 risk estimates to the "direct" estimates of UNSCEAR 1982 which tabulates genetic risk separately by the direct method and by the doubling dose method. The risk estimated by the direct method does not include the same types of damage estimated by doubling doses and was not considered further. Moreover, the BEIR-3 genetic risk estimates provide a better estimate of uncertainty than the UNSCEAR 1982 and ICRPTG estimates because the BEIR-3 Committee assigned a range of uncertainty for multifactorial diseases (>5 percent to <50 percent) which reflects the uncertainty in the numbers better than the other estimates do (5 percent and 10 percent, respectively).

In developing the average mutation rate for the two sexes used in the calculation of the relative mutation risk, the BEIR-3 Committee postulated that the induced mutation rate in females was about 40 percent of that in males (NAS80). Recent studies by Dobson et al. suggest that the assumption was invalid and that human oocytes should have a risk equivalent to that of human spermatogonia. This would increase the risk estimate obtained from doubling-dose methods by a factor of 1.43 (Do83a, Do83b, Do84a, Do84b).

We recognize, however, that the use of the doubling-dose concept does assume that radiation-induced genetic damage is in some way proportional to "spontaneous" damage. As noted earlier, the recent evidence obtained in insects (Drosophila) and bacteria (E. coli) supports the hypothesis that, with the exception of "hot spots" for mutation, the radiation-induced mutation rate is proportional to the spontaneous rate (UNSCEAR82). No proof that this is also true in mammals is available yet.

The BEIR-3 estimates give a considerable range. To express the range as a single estimate, the geometric mean of the range is used, a method first recommended by UNSCEAR (UNSCEAR58) for purposes of calculating genetic risk. The factor of 3 increase in risk for high dose rate, low-LET radiation noted earlier is also used.

The question of RBE for high-LET radiation is more difficult. As noted above, estimated RBEs for plutonium-239 alphas versus chronic gamma radiation for reciprocal translocations as determined by cytogenetic analyses are between 23 and 50 (NAS80, UNSCEAR82). However, the observed RBE for single locus mutations in developing offspring of male mice given plutonium-239 compared to those given X-ray irradiation is 4 (NAS80). The average of RBEs for these reciprocal translocations for specific locus mutations is 20.25. Since reported neutron RBEs are similar to those listed above for plutonium-239 alpha radiation, we use an RBE of 20 to estimate genetic risks for all high-LET radiations. This is consistent with the RBE for high-LET particles recommended for estimated genetic risks associated with space flight (Gr83b).

Genetic risk estimates used by EPA for high- and low-LET radiations are listed in Table G-12. As noted earlier, EPA uses the dose received before age 30 in assessing genetic risks.

The EPA estimates in Table G-12 are limited, like all other human genetic risk estimates, by the lack of confirming evidence of genetic effects in humans. These estimates depend on a presumed resemblance of radiation effects in animals to those in humans. The magnitude of the possible error is indeterminable. The study with the largest data base, the Japanese A-bomb survivors, appears, at best, to provide only an estimate of the minimum doubling dose which calculates the maximum estimate of genetic risk in man. However, doubling-dose estimates are also uncertain because the number of human disorders having a recognized genetic component is constantly increasing, and the type of genetic damage implicated in a specific disorder may change. The combined uncertainties in doubling dose estimates and the magnitude of genetic contributions to various disorders probably introduce an overall uncertainty of about an order of magnitude in the risk estimates. Moreover, the BEIR Committee in deriving its estimate has assumed that almost all of the risk was due to recessive mutations which would eventually be eliminated. To what extent this occurs will depend on medical practices in the future. It is possible, as our knowledge of medicine improves, that recessive hereditary defects will be carried on for many more generations than assumed by the BEIR Committee.

Table G-12. Estimated frequency of genetic disorders in a birth cohort due to exposure of the parents to 1 rad per generation

Radiation	Serious Heritable Disorders (Cases per 10^6 liveborn)			
	First Generation		All Generations	
	low[a]	high[b]	low[a]	high[b]
Low Dose Rate, Low-LET	20	30	260	370
High Dose Rate, Low-LET	60	90	780	1110
High-LET	400	600	5200	7400

[a] Female sensitivity to induction of genetic effects is 40 percent as great as that of males.

[b] Female sensitivity to induction of genetic effects is equal to that of males.

The relative risk of high-LET radiation compared to low-dose-rate, low-LET radiation (RBE) is also uncertain. The data are sparse, and different studies often used different endpoints. In addition, the microscopic dosimetry, i.e., the actual absorbed dose in the cells at risk, is poorly known. However, the RBE estimate used by EPA should be within a factor of 5 of the true RBE for high-LET radiation.

G.5.6 Teratogenic Effects

Although human teratogenesis (congenital abnormalities or defects) associated with x-ray exposure has a long history, the early literature deals mostly with case reports. Stettner reported a case in 1921 (St21) and Murphy and Goldstein studied a series of pregnancies in which 18 of the children born to 76 irradiated mothers were microcephalic (Mu29, Go29). However, the irradiation exposures were high.

In 1930, Murphy exposed some rats to X-rays at doses of 200 R to 1600 R. Thirty-four of 120 exposed females had litters, and 5 of the litters had animals with developmental defects (Mu30). He felt that this study confirmed his clinical observations and earlier reports of animal studies. Although there were additional studies of radiation- induced mammalian.teratogenesis before 1950, the majority of the studies were done after that time (see Ru53 for a review), perhaps reflecting interest in radiation hazards caused by the explosion of nuclear weapons in 1945 (Ja70).

Much of the work done after World War II was done in mice (Ru50, Ru54, Ru56) and rats (Wi54, Hi54). Early studies, at relatively high radiation exposures, 25 R and above, established some dose response relationships. More importantly, they established the time table of sensitivity of the developing rodent embryo and fetus to radiation effects (Ru54, Hi53, Se69, Hi66).

Rugh, in his review of radiation teratogenesis (Ru70) listed the reported mammalian anomalies and the exposure causing them. The lowest reported exposure was 12.5 R for structural defects and 1 R for functional defects. He also suggested human exposure between ovulation and about 7 weeks gestational age could lead to structural defects and from about 6 weeks gestational age until birth could lead to functional defects. In a later review (Ru71), he suggested structural defects in the skeleton might be induced as late as the 10th week of gestation and functional defects as early as the 4th week. It should be noted that the gestation period in mice is much shorter than that in humans and that weeks of gestation referred to above are in terms of equivalent stages of mouse-human development. Estimates of equivalent gestational age are not very accurate.

In the reports of animal studies it appeared as if teratologic effects, other than perhaps growth retardation, had a threshold for induction of effects (Ru54, Ru53, Wi54). However, Ohzu (Oh65) showed that doses as low as 5 R to preimplantation mouse embryos caused increased resorption of implanted embryos and structural abnormalities in survivors. Then in 1970, Jacobsen (Ja70) reported a study in which mice were exposed to 5, 20 or 100 R on the 8th day of pregnancy. He concluded that the dose-response function for induction of skeletal effects was linear, or nearly linear, with no observable threshold. This appears consistent with a report by Russell (Ru57), which suggested a threshold for some effects whereas others appeared linear.

Rugh (Ru71) suggested there may be no threshold for radiation-induced congenital effects in the early human fetus. In the case of microcephaly (small head size) and mental retardation, at least this may be the case. For other teratogenic effects, the dose response in humans is unknown. In 1978, Michel and Fritz-Niggli (Mi78) reported induction of a significant increase in growth retardation, eye and nervous system abnormalities, and post implantation losses in mice exposed to 1 R. The increase was still greater if there was concurrent exposure to radiosensitizing chemicals such as iodoacetimide or tetracycline (Mi78).

One of the problems with the teratologic studies in animals is the difficulty of determining how dose response data should be interpreted.

Russell (Ru54) pointed out some aspects of the problem: 1) although radiation is absorbed throughout the embryo, it causes selective damage which is consistently dependent on the stage of embryonic development at the time of irradiation, and 2) the damaged parts respond, in a consistent manner, within a narrow time range. However, while low dose irradiation at a certain stage of development produces changes only in components at their peak sensitivity, higher doses may induce additional abnormalities which have peak sensitivity at other stages of development, and may further modify expression of the changes induced in parts of the embryo at peak sensitivity during the time of irradiation. In the first case, damage may be to primordial cells themselves, while in the second, the damage may lead indirectly to the same or different endpoints.

The embryo/fetus starts as a single fertilized egg and divides and differentiates to produce the normal infant at term. (The embryonic period, when organs develop, is the period from conception through 7 weeks gestational age. The fetal period, a time of *in utero* growth, is the period from 8 weeks gestational age to birth.) The different organ and tissue primordia develop independently and at different rates. However, they are in contact through chemical induction or evaporation (Ar54). These chemical messages between cells are important in bringing about orderly development and the correct timing and fitting together of parts of organs or organisms. While radiation can disrupt this pattern, interpretation of the response may be difficult. Since the cells in the embryo/fetus differentiate, divide, and proliferate at different times during gestation and at different rates, gestational times when cells of specific organs or tissues reach maximum sensitivity to radiation are different. Each embryo/fetus has a different timetable. In fact, each half (left/right) of an embryo/fetus may have a slightly different timetable.

In addition, there is a continuum of variation from the hypothetical normal to the extreme deviant, which is obviously recognizable. There is no logical place to draw a line of separation between normal and abnormal. The distinction between minor variations of normal and frank malformation, therefore, is an arbitrary one, and each investigator must establish his own criteria and apply them to spontaneous and induced abnormalities alike (HWC73). For example, some classify mental retardation as IQ 80 or lower, some classify on ability to converse or hold a job, some on the basis of the need to be institutionalized.

Because of the problems in interpretation listed above, it appears a pragmatic approach is useful. The dose response should be given as the simplest function that fits the data, often linear or linear with a threshold. No attempt should be made to develop complex dose-response models unless the evidence is unequivocal.

The first report of congenital abnormalities in children exposed *in utero* to radiation from atomic bombs was that of Plummer (P152). In this selected group, twelve children with microcephaly of which 10 also had mental retardation had been identified in Hiroshima in the *in utero* exposed survivors. They were found as part of a program started in 1950 to study children exposed in the first trimester of gestation. In 1955 the program was expanded to include all survivors exposed *in utero*.

Studies initiated during the program have shown radiation related 1) growth retardation; 2) increased microcephaly; 3) increased mortality, especially infant mortality; 4) temporary suppression of antibody production against influenza; and 5) increased frequency of chromosomal aberrations in peripheral lymphocytes (Ka73).

Although there have been a number of studies of Japanese A-bomb survivors, including one showing a dose and gestational age related increase in postnatal mortality (Ka73), only incidence of microcephaly and mental retardation have been investigated to any great extent. In the most recent report, Otake and Schull (Ot83) showed that mental retardation was associated with exposure between 8 and 15 weeks of gestation (10 to 17 weeks of gestation if counted from the last menstrual period). They further found a linear dose-response relationship for induction of mental retardation that had a slope yielding a tentative estimate of doubling dose for mental retardation of about 2 rad, fetal-absorbed dose (Ot83). Classification as mentally retarded was based on "unable to perform simple calculations, to care for himself or herself, or if he or she was completely unmanageable or had been institutionalized" (Ot83).

Estimates of the risk of mental retardation for a rad of embryo/fetus exposure in the U.S. population can be derived by three methods. The first and easiest method is to use the absolute risk calculated by Otake and Schull for the Japanese survivors (Ot84). A second method is to use the doubling dose calculated by Otake and Schull (Ot83) times the incidence of mental retardation per 10^3 live births. Unfortunately, a number of assumptions must be made to establish the incidence of mental retardation per 10^3 live births. Mental retardation may be classified as mild (IQ 50-70), moderate (IQ 35-49), severe (IQ 20-34) and profound (IQ <20) (WHO75). However, some investigators use only mild mental retardation (IQ 50-70) and severe mental retardation (IQ <50) as classes (Ha81, St84). Mental retardation is not usually diagnosed at birth but at some later time, often at school age. Since the mental retardation may have been caused before or during gestation, at the time of birth, or at some time after birth, that fraction caused before or during gestation must be estimated. In like manner since mental retardation caused before birth may be due to genetic conditions, infections, physiologic conditions, etc., the fraction related to unknown causes during gestation must be estimated. This is the fraction that might possibly be doubled by radiation exposure.

A third method to estimate the risk is indirectly using the relationship of microcephaly and mental retardation reported in the Japanese survivors (Wo65, Ot83). If head size is assumed to be normally distributed, then the fraction of the population with a head size 2 or 3 standard deviations smaller than average can be obtained from statistical tables. The fraction of 10^3 liveborn with microcephaly multiplied by the proportion of mental retardation associated with that head size yields an estimate of the incidence of mental retardation per 10^3 live births, which can then be used with the doubling dose to estimate the risk as described above.

Risk estimates for mental retardation are derived below for comparison purposes using each of the three methods described above.

A. Estimate of Incidence Per Rad Based on Direct Application of the Slope of the Japanese Data

Otake and Schull (Ot84) gave an estimate of 'The Relationship of Mental Retardation to Absorbed Fetal Exposure in the "Sensitive" Period When All "Controls" are Combined.' The estimate of 0.416 cases of mental retardation per 100 rad could be directly applicable to a U.S. population. In this case the risk estimate would be about:

4 cases of mental retardation per rad per 1000 live births.

B. Estimate of Incidence Per Rad Based on the Doubling Dose

The Otake and Schull report (Ot83) suggested the doubling dose for mental retardation was about 2 rad fetal absorbed dose or about a 50 percent increase in mental retardation per rad. It would seem reasonable that this doubling dose would apply only to ideopathic cases of mental retardation caused during gestation. That is, those which have no known genetic, viral, bacterial, etc., cause.

Data from studies of the prevalence of mental retardation in school age populations in developed countries suggest a prevalence of 2.8 cases/1000 (Uppsala County, Sweden) to 7.4 cases/1000 (Amsterdam, Holland) of severe mental retardation, with a mean of about 4.3 ± 1.3 cases/1000 (St84). Where data are available for males and females separately, the male rate is about 30 percent higher than the female rate (St84). Historically, the prevalence of mild mental retardation has been 6 to 10 times greater than that of severe mental retardation. But in recent Swedish studies, the rates of prevalence of mild and severe mental retardation have been similar (St84). This was suggested to be due to a decline in the "cultural-familial syndrome." That is, improved nutrition, decline in infection and diseases of childhood, increased social and intellectual stimulation, etc., combined to reduce the proportion of nonorganic mental retardation and, therefore, the prevalence of mild mental retardation (St84).

In studies of the causes of mental retardation, 23 percent to 42 percent of the mental retardation has no identified cause (Gu77, Ha81, St84). It is this portion of the mental retardation which may be susceptible to increase from radiation exposure of the embryo/fetus. In that case, the prevalence of ideopathic mental retardation would be 0.6 to 3.1 cases per 1000 of severe mental retardation and perhaps an equal number of cases of mild mental retardation.

For purposes of estimating the effects of radiation exposure of the embryo/fetus, a risk of spontaneous ideopathic mental retardation of 1 to 6 per 1000 will be used. If this spontaneous ideopathic mental retardation can be increased by radiation the estimate would be:

(1 to 6 cases per 1000 live births)(0.5 increase per rad)

or about 0.5 to 3 cases of mental retardation per rad per 1000 live births.

This estimate may be biased low because mental retardation induced during gestation is often associated with high childhood death rate (St84). If this is generally true for ideopathic causes of mental retardation, it would cause an underestimation of the risk.

C. Estimate of Incidence Per Rad Based on Incidence of Microcephaly

1) Of live born children, 2.275 percent will have a head circumference 2 standard deviations or more smaller than average, 0.621 percent will have a head circumference 2.5 standard deviations or more smaller than average, and 0.135 percent will have a head circumference 3 standard deviations or more smaller than average (statistical estimate based on a normal distribution).

2) There is evidence in a nonselected group of 9,379 children that mental retardation can be estimated using incidence of microcephaly, even though head circumference in the absence of other supporting data, e.g., height or proportion, is an uncertain indicator of mental retardation. Based on a study of 9,379 children, Nelson and Deutschberger (Ne70) concluded that about half of the children with a head circumference 2.5 standard deviations or more smaller than average had IQs of 79 or lower. Since 0.67 percent of those studied were in this group, the observed number is about what would be expected based on the normal distribution of head size in a population, 0.62 percent. The estimated incidence of mental retardation per live birth in a population would be:

$$(6.7 \text{ cases of microcephaly per 1000 live births}) \times$$

$$0.5 \ \frac{\text{cases of mental retardation}}{\text{case of microcephaly}}$$

or about 3.4 cases of mental retardation per 1000 live births.

3) A first approximation of risk of mental retardation might then be:

(3.4 cases of mental retardation per 1000 live births)

(0.5 increase per rad)

or about 2 cases of mental retardation per 1000 live births per rad.

Both microcephaly and mental retardation were increased in Japanese survivors (Wo65, Wo66). However, the relationship of mental retardation to microcephaly in Japanese A-bomb survivor data is not clear. Estimates range from about half of those with head sizes 2 or more standard deviations smaller than average having mental retardation (RERF78), a result similar to that observed by Nelson and Deutschberger (Ne70), to about ten percent of those with small head size having mental retardation (Wo66). Therefore, the above estimate based on the incidence of microcephaly in a population should be a reasonable estimate of the risk from radiation.

Summary of the Calculated Risk of Mental Retardation

The risk of increased mental retardation per rad of embryo/fetus exposure during the 8- to 15-week gestational period estimated above ranges from about 5×10^{-4} to 4×10^{-3} cases per live birth, the largest being a direct estimate. The geometric mean of these estimates is 1.4×10^{-3}; the arithmetic mean is 2.4×10^{-3} cases per live birth.

All the estimates derived above by any of the three methods are in the same range as an earlier UNSCEAR (UNSCEAR77) estimate of an increase of 1×10^{-3} cases of mental retardation per rad per live birth. The UNSCEAR estimate, however, did not consider gestational age at the time of exposure. The Otake and Schull report (Ot83) did address gestational age and estimated a higher risk, but a narrower window of susceptibility.

If the estimates are applicable, the 15 mrad of low-LET background radiation delivered during the 8- to 15-week gestational age-sensitive period could induce a risk of 6×10^{-5} to 7.5×10^{-6} cases of mental retardation per live birth. This can be compared to an estimate of a spontaneous occurrence of 1.5×10^{-2} to 3.4×10^{-3} cases of mental retardation per live birth.

Japanese A-bomb survivors exposed in utero also showed a number of structural abnormalities and, particularly in those who were microcephalic, retarded growth (Wo65). No estimate has been made of the radiation-related incidence or dose-response relationships for these abnormalities. However, UNSCEAR (UNSCEAR77) made a very tentative estimate based on animal studies that the increased incidence of structural abnormalities in animals may be 5×10^{-3} cases per R per live born, but stated that projection to humans was unwarranted. In any event, the available human data cannot show whether the risk estimates derived from high dose animal data overestimates the risk in humans.

It should be noted that all of the above estimates are based on high dose rate low-LET exposure. UNSCEAR in 1977 also investigated the dose-rate question and stated:

> "In conclusion, the majority of the data available for most species indicate a decrease of the cellular and malformature effects by lowering the dose rate or by fractionating the dose. However, deviations from this trend have been well documented in a few instances and are not inconsistent with the knowledge about mechanisms of the teratogenic effects. It is therefore impossible to assume that dose rate and fractionation factors have the same influence on all teratological effects." (UNSCEAR77).

From this analysis, EPA has concluded that a range of risk is 4×10^{-3} to 5×10^{-4} cases of mental retardation per live birth per rad of low-LET radiation delivered between weeks 8 and 15 of gestation with no threshold identified at this time.

No attempt can be made now to estimate total teratogenic effects. However, it should be noted that the 1977 UNSCEAR estimate from animals was 5×10^{-3} cases of structural abnormalities per R per live birth (about the same number per rad of low-LET). This estimate must be viewed as a minimum one since it is based, to a large extent, on observation of grossly visible malformations. Differences in criteria for identifying malformations have compounded the problem, and questions of threshold and species differences have made risk projection to humans unwarranted.

G.5.7 Nonstochastic Effects

Nonstochastic effects, those effects that increase in severity with increasing dose and may have a threshold, have been reviewed in the 1982 UNSCEAR report (UNSCEAR82). In general, acute doses of 10 rad low-LET radiation and higher are required to induce these effects. It is possible that some of the observed effects of *in utero* exposure are nonstochastic, e.g., the risk of embryonic loss, estimated to be 10^{-2} per R (UNSCEAR77), following radiation exposure soon after fertilization. However, there are no data to address the question. Usually, no non-stochastic effects of radiation are expected at environmental levels of radiation exposure.

G.6 Radiation Risk - A Perspective

To provide a perspective on the risk of fatal radiogenic cancers and the hereditary damage due to radiation, we have calculated the risk from background radiation to the U.S. population using the risk coefficients presented in this chapter and the computer codes described in Appendix H.

The risk resulting from background radiation is a useful perspective for the risks caused by releases of radionuclides. Unlike cigarette smoking, auto accidents, and other measures of common risks, the risks resulting from background radiation are neither voluntary nor the result of alcohol abuse. The risk caused by background radiation is very largely unavoidable; therefore, it is a good benchmark for judging the estimated risks from radionuclide releases. Moreover, to the degree that the estimated risk of radionuclides is biased, the same bias is present in the risk estimates for background radiation.

Low-LET background radiation has three major components: cosmic radiation, which averages to about 28 mrad per year in the U.S.: terrestrial sources, such as radium in soil, which contributes an average of 26 mrad per year (NCRP75); and the low-LET dose resulting from internal emitters. The last differs between organs, to some extent, but for soft tissues is about 24 mrad per year (NCRP75). Fallout from nuclear weapons tests, naturally occurring radioactive materials in buildings, etc., contributes about another 10 mrem for a total low-LET whole-body dose of about 90 mrad per year. The lung and bone receive somewhat larger doses due to high-LET radiations; see below. Although extremes do occur, the distribution of this background annual dose to the U.S. population is relatively narrow. A population weighted analysis indicates that 80 percent of the U.S. population would receive annual doses that are between 75 mrad per year and 115 millrad per year (EPA81).

As outlined in Section G.2, the BEIR-3 linear models yield, for lifetime exposure to low-LET radiation, an average lifetime risk of fatal radiogenic cancer of 280 per 10^6 person rad. Note that this average is for a group having the age- and sex-specific mortality rates of the 1970 U.S. population. We can use this datum to calculate the average lifetime risk due to low-LET background radiation as follows. The average duration of exposure in this group is 70.7 years and at 9×10^{-2} rad per year, the average lifetime dose is 6.36 rad. The risk of fatal cancer per person in this group is:

$$\frac{\text{280 fatalities}}{10^6 \text{ person rad}} \times 6.36 \text{ rem} = 1.78 \times 10^{-3}$$

or about 0.18 percent of all deaths. The vital statistics we use in our radiation risk analyses indicate that the probability of dying from cancer in the United States from all causes is about 0.16, i.e., 16 percent. Thus, the 0.18 percent result for the BEIR-3 linear dose response model indicates that about 1 percent of all U.S. cancer is due to low-LET background radiation. The BEIR-3 linear quadratic model indicates that about 0.07 percent of all deaths are due to low-LET background radiation or about 0.4 percent of all cancer deaths.

Table G-6 indicates a risk of 460 fatalities per 10^6 organ rad for alpha emitters in lung tissue. The lifetime cancer from this exposure is:

$$\frac{460 \text{ fatalities}}{10^6 \text{ organ rad}} \times \frac{0.03 \text{ rad}}{\text{year}} \times 70.7\text{years} = 0.98 \times 10^{-3}$$

This is twice the risk due to low-LET background radiation calculated by means of the BEIR-3 linear quadratic model and more than half of the risk calculated by means of the BEIR-3 linear model.

The 1982 UNSCEAR report indicates that the average annual dose to the endosteal surfaces of bone due to naturally-occurring high-LET alpha radiation is about 6 mrad per year or, for a quality factor 20, 120 mrem per year (UNSCEAR82). Table G-6 indicates that the lifetime risk of fatal bone cancer due to this portion of the naturally occurring radiation background is:

$$\frac{20 \text{ cases}}{10^6 \text{ person rad}} \times \frac{0.006 \text{ rad}}{\text{year}} \times 70.7 \text{ years} = 8.5 \times 10^{-6}.$$

The spontaneous incidence of serious congenital and genetic abnormalities has been estimated to be about 105,000 per 10^6 live births, about 10.5 percent of live births (NAS80, UNSCEAR82). The low-LET background radiation dose of about 90 mrad/year in soft tissue results in a genetically significant dose of 2.7 rad during the 30-year reproductive generation. Since this dose would have occurred in a large number of generations, the genetic effects of the radiation exposure are thought to be an equilibrium level of expression. Since genetic risk estimates vary by a factor of 20 or more, EPA uses a log mean of this range to obtain an average value for estimating genetic risk. Based on this average value, the background radiation causes 700 to 1000 genetic effects per 10^6 live births, depending on whether or not the oocyte is as sensitive to radiation as the spermatogonia (see Section G.5). This result indicates that about 0.67 percent to 0.95 percent of the current spontaneous incidence of serious congenital and genetic abnormalities may be due to the low-LET background radiation.

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APPENDIX H: A DESCRIPTION OF THE RADRISK AND CAIRD COMPUTER CODES USED BY EPA IN ASSESSING DOSES AND RISKS FROM RADIATION EXPOSURE

H.1 Introduction

This appendix provides a brief overview of the RADRISK (Du80) and CAIRD (Co78) computer codes used by the Environmental Protection Agency to assess the health risk from radiation exposures. It describes how the basic dose calculations are performed and describes the mechanics of the life table implementation of the risk estimates derived in Appendix G.

H.2 Overview of the EPA Analysis

RADRISK, the computer code used to calculate dose and risk (Du84, Su81, Du80), calculates the radiation dose and risk resulting from an annual unit intake of a given radionuclide or the risk resulting from external exposure to a unit concentration of radionuclide in air or on ground surface. Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the exposure associated with a specific source.

As outlined in Appendix F, estimates of the annual dose rate to organs and tissues of interest are calculated by using, primarily, models recommended by the International Commission on Radiological Protection (ICRP79, ICRP80). Because EPA usually considers lifetime exposures to a general population, these dose rates are used in conjunction with a life table analysis of the increased risk of cancer resulting from radiation (Co78). This analysis, described below, takes account of competing risks and the age of the population at risk.

H.3 Dose Rates from Internal Exposures

Internal exposures occur when radioactive material is inhaled or ingested. The RADRISK code implements contemporary dosimetric models to estimate the dose rates at various times to specified reference organs in the body from inhaled or ingested radionuclides. The dosimetric methods in RADRISK are adapted from those of the INREM-II code (Ki78) which is based on models recommended by the International Commission on Radiological Protection (ICRP79). The principal qualitative difference is that RADRISK computes dose rates to specified organs separately for high and low linear energy transfer (LET) radiations, while INREM-II calculates the committed dose equivalent to specified organs. The time-dependent dose rates are used in the life table calculations of RADRISK.

In RADRISK, the direct intake of each radionuclide is treated separately. For decay chains, the ingrowth and dynamics of decay products (daughters) in the body after intake of a parent radionuclide are considered explicitly in the calculation of dose rate. The decay product contributions to the dose rate are included in the dose calculations, based on the metabolic properties of the element and the organ in which they occur.

The dose rate $\dot{D}_i(X,t)$ to target organ X at time t due to radionuclide i $(1 \le i \le N)$ residing in organs $Y_1, Y_2, \ldots, Y_m$ is a measure of the energy deposited annually in a given mass of tissue as a result of radioactive decay, and is computed as:

$$\dot{D}_i(X;t) = \sum_{k=1}^{m} \dot{D}_i(X \leftarrow Y_k;t) \qquad \text{(H-1)}$$

where

$$\dot{D}_i(X \leftarrow Y_k;t) = S_i(X \leftarrow Y_k)A_{ik}(t) \qquad \text{(H-2)}$$

$A_{ik}(t)$ = activity of radionuclide i in organ Y_k at time t measured from the initial intake of i into the body,

$S_i(X \leftarrow Y_k)$ = average dose rate to target organ X per unit activity of the radionuclide i uniformly distributed in source organ Y_k (Sn74, Du80).

The summation is taken over all source organs Y. Implicit in the definitions is the assumption of uniform distribution of activity of radionuclide i in each source organ, as is the assumption of averaging the dose rate over the mass of the target organ. Although estimates of dose to an organ include contributions from activity distributed throughout the body (for penetrating radiations), activity within that organ generally contributes the principal component of dose [i.e., $\dot{D}_i(X \leftarrow X;t)$ is the principal component of $\dot{D}_i(X;t)$].

The time rate of change of activity in the body is modeled by a system of ordinary differential equations, with each equation describing the rate of change of activity in a conceptual compartment of the body. For radionuclides that are part of a decay series, there may be formation of radioactive daughters in a given compartment that have different chemical and physical properties from those of the parent. Unlike the models given in ICRP80, the specific metabolic properties of the daughter are evaluated when they differ from those of the parent. This refinement is under active consideration by ICRP experts. In almost all cases, doses to soft tissues calculated on this basis differ only slightly, if at all, from ICRP80 dose estimates, but the difference is large for some radionuclides when the parent is incorporated into bone, as in lead-210. For this radionuclide the ICRP80 model has been used without any modifications.

A schematic representation of radioactivity movement in the body is shown in Appendix F. Except for radon daughters, which are considered separately, inhaled activity is assumed to be originally deposited in the lungs (distributed among the nasopharyngeal, tracheobronchial, and pulmonary regions), and ingested activity is originally deposited in the stomach. From the lungs, activity may be absorbed by the bloodstream or migrate to the stomach. Activity in the stomach may proceed through the small intestine, upper large intestine, and lower large intestine; activity may be absorbed by the bloodstream from any of these four segments, although only absorption from the small intestine is considered in this study.

The activity, $A_{ik}(t)$, of radionuclide i in organ k may be divided among several "pools" or "compartments," denoted here by the subscript p. Each differential equation describing the rate of change of activity within a compartment is a special case of the equation:

$$\dot{A}_{ipk} = -(\lambda_i^R + \lambda_{ipk}^B)A_{ipk} + c_{ipk}(\lambda_i^R \sum_{j=1}^{i-1} B_{ij} \sum_{r=1}^{L_{jk}} A_{jr} + P_{ik}), \quad p=1,\ldots, p_{ik} \qquad \text{(H-3)}$$

where

$\dot{A}_{ipk}$ = activity of radionuclide i in compartment p of organ k,

L_{ipk} = number of exponential terms in the retention function for radionuclide i in organ k,

B_{ij} = branching ratio of radionuclide j to nuclide i,

λ_i^R = rate coefficient (time^{-1}) for radiological decay of radionuclide i,

λ_{ipk}^B = rate coefficient (time^{-1}) for biological removal of radionuclide i from compartment p of organ k,

c_{ipk} = fractional coefficient for radionuclide i in the p-th compartment of organ k,

P_{ik} = inflow rate of radionuclide i into organ k.

If the inflow rate P_{jk} remains constant, the equations may be solved explicitly for $A_{jk}(t)$ as described by Killough, Dunning, and Pleasant (Ki78). In many cases the inflow into a compartment will not be a constant rate over a long period of time. To handle this problem, the time interval over which solution of the activity equation is desired (e.g., 110 years) is divided into 1-year subintervals. The inflow rate on each subinterval is then taken to be that constant value which would yield the total activity flowing out of the preceding compartment(s) during the same subinterval.

The model used in RADRISK for particulate deposition and retention in the respiratory tract is the ICRP task group lung model (Mo66, ICRP72). In this model, shown in Appendix F, there are four major regions: the naso-pharyngeal, tracheobronchial, pulmonary, and lymphatic tissues. A fraction of the inhaled activity is initially deposited in each of the naso-pharyngeal, tracheobronchial, and pulmonary regions. The material is then cleared (removed) from the lung to the blood and the gastrointestinal tract, also as shown in Appendix F. Deposition and clearance of inspired particulates in the lung are controlled by the particle size and solubility classification.

The size distribution of the particles is specified by the activity median aerodynamic diameter (AMAD); where no AMAD is known, a value of 1.0 micron is assumed. The model employs three solubility classes, based on the chemical properties of the radionuclide; classes D, W, and Y correspond to rapid (days), intermediate (weeks), and slow (years) clearance, respectively, of material deposited in the respiratory passages. Inhaled nonreactive, i.e., noble, gases are handled as a special case.

Movement of activity through the gastrointestinal (GI) tract is simulated with a catenary model, consisting of four segments: stomach, small intestine, upper large intestine, and lower large intestine. Exponential outflow of activity from each segment into the next or out of the system is assumed. Outflow rate constants are calculated from the transit times of Eve (Ev66). Although absorption may occur from any combination of the four segments, only activity absorbed into the blood from the small intestine is normally considered; the fractional absorption from the small intestine into the blood is traditionally denoted f_1.

Activity absorbed by the blood from the GI or respiratory tract is assumed to be distributed immediately to systemic organs. The distribution of activity to these organs is specified by fractional uptake coefficients. The list of organs in which activity is explicitly distributed (termed source organs) is element-dependent, and may include such organs as bone or liver where sufficient metabolic data are available. This list is complemented by an additional source region denoted as OTHER, which accounts for that systemic activity not distributed among the explicit source organs; uniform distribution of this remaining activity within OTHER is assumed.

Radioactive material that enters an organ may be removed by both radioactive decay and biological removal processes. For each source organ, the fraction of the initial activity remaining at any time after intake is described by a retention function consisting of one or more exponentially decaying terms.

The metabolic models and parameters employed in the present study have been described by Sullivan et al. (Su81). In most cases, the models are similar or identical to those recently recommended by the ICRP (ICRP79, ICRP80, ICRP81). However, some differences in model parameters do exist for some radionuclides (Su81). In particular, parameter values that are thought to be more representative of metabolism following low-level environmental exposures, rather than occupational exposures, have been used in this analysis [e.g., f_1=0.2 for uranium in the environment (ICRP79, NAS83)]. For transuranic isotopes, metabolic parameters from EPA77, related comments from EPA78 and from the National Radiological Protection Board (Ha82), have been used rather than those from ICRP80. These parameters are listed in Table H-1.

The EPA values were recommended by U.S. experts on transuranic element metabolism at Battelle Pacific Northwest Laboratory (EPA78). The recently-adopted National Radiation Protection Board f_1 values for transuranics in the general environment are closer to the values proposed by EPA in 1977 than those currently advocated by ICRP for occupational exposures. The larger f_1 values will increase the estimated dose and risk from ingestion of transuranic materials but have little effect on doses following inhalation.

H.4 Dose Rates from External Exposures

Because of the penetrating nature of photons, radioactivity need not be taken into the body to deliver a dose to body organs. Energy absorbed from photons emitted by radionuclides in the air or on the ground surface may also contribute to the overall risk. Natural background radiation is an example of an important external exposure, ordinarily contributing the largest component of dose to people.

TABLE H-1:
Small intestine to blood transfer fractions, f_1, for transuranic elements

Element Isotope	EPA Child 0-12 mo	EPA Adult >12 mo	NRPB Adult	NRPB Child 0-12 mo	NRPB Child 0-3 mo
Plutonium-238 and 241					
Oxide form	10^{-2}	10^{-3}	10^{-5}(b)	5×10^{-4}(b)	10^{-3}(b)
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.(a)	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Plutonium-239 and 240					
Oxide form	10^{-3}	10^{-4}	10^{-5}(b)	5×10^{-4}(b)	10^{-3}(b)
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Americium					
Oxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Curium					
Oxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Nonoxide form	10^{-2}	10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Bio. inc.	5×10^{-2}	5×10^{-3}	5×10^{-4}	5×10^{-3}	10^{-2}
Neptunium		10^{-3}	10^{-3}	5×10^{-3}	10^{-2}

(a) Biologically incorporated form.
(b) Hydroxide form.

Source: EPA77, EPA78, Ha82.
NRPB: National Radiological Protection Board.

Organ dose rates to an individual immersed in contaminated air or standing on a contaminated ground surface are computed by Kocher's DOSFACTER computer code (Ko81). These calculations assume that the radionuclide concentration is uniform throughout an infinite volume of air or area of ground surface, and that the exposed individual is standing on the ground surface. Only photons penetrate the body sufficiently to deliver a significant dose to internal organs, and only doses from photon radiation are considered in this analysis. Beta radiation is far less penetrating and delivers a dose only to the body surface; because skin is not a target tissue of concern in this analysis, no consideration of beta contributions to dose is required. Alpha particles have even less penetration ability, and are also excluded from consideration here.

The photon dose rate factor $\dot{D}_i^\gamma(X)$ for a given target organ, X, of an individual immersed in contaminated air at any time may be expressed as:

$$\dot{D}_i^\gamma(X) = cK_{pm} \frac{1}{\rho_a} \sum_n f_n^\gamma E_n^\gamma \left[\frac{(\mu/\rho)_t}{(\mu/\rho)_a} G^X \right]_n \qquad \text{(H-4)}$$

where

ρ_a = density of air,

K_{pm} = 0.5 = particle-medium correction factor,

f_n^γ = intensity of nth discrete photon (number/disintegration),

E_n^γ = energy of nth photon,

μ/ρ = photon mass energy absorption coefficient, with subscripts "t" and "a" denoting tissue and air, respectively, for photons of energy E_n,

G^X = ratio of absorbed dose in organ X to absorbed dose at the body surface.

c = unit conversion proportionality contrast.

The terms μ/ρ and G^X are functions of photon energy, E_n^γ.

The photon dose rate factor $\dot{D}^{\gamma}_{iz}$ (X) to organ X of an individual at a distance z above a unit concentration contaminated ground surface may be computed as:

$$\dot{D}^{\gamma}_{iz}\ (X) = 0.5cK_{pm} \sum_{n} f^{\gamma}_{n} E^{\gamma}_{n}[(\mu/\rho)_{t}]n \left[\int_{z}^{\infty} 1/r \exp(-\mu_{an}r)\ dr - [C_{an}/(\dot{D}_{an}-1)] \exp[(\dot{D}_{an}-1)\mu_{an}z]\right] G^{X} \qquad \text{(H-5)}$$

where

K_{pm} = 1.0 = particle-material correction factor,

μ_{an} = mass attenuation coefficient for the nth discrete proton,

z = height of reference position above ground surface (taken to be 1 meter in this study).

c = unit conversion proportionality constant.

The coefficients C_{an} and D_{an} are functions of the photon energy. For detailed discussion of the derivation of these equations and a tabulation of dose rate factors for various radionuclides, see Kocher (Ko79, Ko81).

In the analysis here, the dose rate factors described by these equations are scaled to achieve a continuous exposure of 1 pCi/cm^3 for air immersion and 1 pCi/cm^2 for ground surface exposure. Risk estimates for these exposure pathways are based on continuous lifetime exposure to these levels.

H.5 Life Table Analysis to Estimate the Risk of Excess Cancer

Radiation effects can be classified as stochastic or nonstochastic (NAS80, ICRP77). For stochastic effects, the probability of occurrence of the effect, as opposed to the severity, is a function of dose; induction of cancer, for example, is considered a stochastic effect. Nonstochastic effects are those health effects for which the severity of the effect is a function of dose; examples of nonstochastic effects include cell killing, suppression of cell division, cataracts, and nonmalignant skin damage.

At the low levels of radiation exposure attributed to radionuclides in the environment, the principal health detriment is the induction of cancers (solid tumors and leukemia), and the expression, in later generations, of genetic effects. In order to estimate these effects, instantaneous dose rates for each organ at specified times are sent to a subroutine adaptation of CAIRD (Co78) contained in the RADRISK code. This subroutine uses annual doses derived from the transmitted dose rates to estimate the number of incremental fatalities in the cohort due to radiation-induced cancer in the reference organ. The calculation of incremental fatalities is based on estimated annual incremental risks, computed from annual doses to the organ, together with radiation risk factors such as those given in the 1980 NAS report BEIR-3 (NAS80). Derivation of the risk factors in current use is discussed in Appendix G.

An important feature of this methodology is the use of actuarial life tables to account for the time dependence of the radiation insult and to allow for competing risks of death in the estimation of risk due to radiation exposure. A life table consists of data describing age-specific mortality rates from all causes of death for a given population. This information is derived from data obtained on actual mortality rates in a real population; mortality data for the U.S. population during the years 1969-1971 (HEW75) are used throughout this study.

The use of life tables in studies of risk due to low-level radiation exposure is important because of the time delay inherent in radiation risk. After a radiation dose is received, there is a minimum induction period of several years (latency period) before a cancer is clinically observed. Following the latency period, the probability of occurrence of a cancer during a given year is assumed to be constant for a specified period, called a plateau period. The length of both the latency and plateau periods depends upon the type of cancer.

During or after radiation exposure, a potential cancer victim may experience years of life in which he is continually exposed to risk of death from causes other than incremental radiation exposure. Hence, some individuals in the population will die from competing causes of death, and are not potential victims of incremental radiation-induced cancer.

Each member of the hypothetical cohort is assumed to be exposed to a specified activity of a given radionuclide. In this analysis each member of the cohort annually inhales or ingests 1 pCi of the radionuclide, or is exposed to a constant external concentration of 1 pCi/cm^3 in air or 1 pCi/cm^2 on ground surfaces. Since the models used in RADRISK are linear, these results may be scaled to evaluate other exposure conditions. The cohort consists of an initial population of 100,000 persons, all of whom are simultaneously liveborn. In the scenario employed here, the radiation exposure is assumed to begin at birth and continue throughout the entire lifetime of each individual.

No member of the cohort lives more than 110 years. The span from 0 to 110 years is divided into nine age intervals, and dose rates to specified organs at the midpoints of the age intervals are used as estimates of the annual dose during the age interval. For a given organ, the incremental probability of death due to radiation-induced cancer is estimated for each year using radiation risk factors and the calculated doses during that year and relevant preceding years. The incremental probabilities of death are used in conjunction with the actuarial life tables to estimate the incremental number of radiation-induced deaths each year.

The estimation of the number of premature deaths proceeds in the following manner. At the beginning of each year, m, there is a probability P^N of dying during that year from nonradiological causes, as calculated from the life table data, and an estimated incremental probability P^R of dying during that year due to radiation-induced cancer of the given organ. In general, for the m-th year, the calculations are:

$M(m)$ = total number of deaths in cohort during year m,
$= [P^N(m) + P^R(m)] \times N(m)$

$Q(m)$ = incremental number of deaths during year m due to radiation-induced cancer of a given organ,
$= P^R(m) \times N(m)$

$N(m+1)$ = number of survivors at the beginning of year m + 1
$= N(m) - M(m)$
$(N(1)=100{,}000)$.

P^R is assumed to be small relative to P^N, an assumption which is reasonable only for low-level exposures (Bu81), such as those considered here. The total number of incremental deaths for the cohort is then obtained by summing Q(m) over all organs for 110 years.

In addition to providing an estimate of the incremental number of deaths, the life table methodology can be used to estimate the total number of years of life lost to those dying of radiation-induced cancer, the average number of years of life lost per incremental mortality, and the decrease in the population's life expectancy. The total number of years of life lost to those dying of radiation-induced cancer is computed as the difference between the total number of years of life lived by the cohort assuming no incremental radiation risk, and the total number of years of life lived by the same cohort assuming the incremental risk from radiation. The decrease in the population's life expectancy can be calculated as the total years of life lost divided by the original cohort size (N(1)=100,000).

Either absolute or relative risk factors can be used. Absolute risk factors, given in terms of deaths per unit dose, are based on the assumption that there is some absolute number of deaths in a population exposed at a given age per unit of dose. Relative risk factors, the percentage increase in the ambient cancer death rate per unit dose, are based on the assumption that the annual rate of radiation-induced excess cancer deaths, due to a specific type of cancer, is proportional to the ambient rate of occurrence of fatal cancers of that type. Either the absolute or the relative risk factor is assumed to apply uniformly during a plateau period, beginning at the end of the latent period.

The estimates of incremental deaths in the cohort from chronic exposure are identically those which are obtained if a corresponding stationary population (i.e., a population in which equal numbers of persons are born and die in each year) is subjected to an acute radiation dose of the same magnitude. Since the total persons years lived by the cohort in this study is approximately 7.07 million, the estimates of incremental mortality in the cohort from chronic irradiation also apply to a one year dose of the same magnitude to a population of this size, age distribution, and age-specific mortality rates. More precise life table estimates for a specific population can be obtained by altering the structure of the cohort to reflect the age distribution of a particular population at risk.

H.6 Risk Analysis Methodology

Risk estimates in current use at EPA are based on the 1980 report (BEIR-3) of the National Academy of Sciences Advisory Committee on the Biological Effects of Ionizing Radiation (NAS80). The form of these risk estimates is, to some extent, dictated by practical considerations, e.g., a desire to limit the number of cases which must be processed for each environmental analysis and a need to conform to limitations of the computer codes in use. For example, rather than analyze male and female populations separately, the risk estimates have been merged for use with the general population; rather than perform both an absolute and a relative risk calculation, average values have been used.

The derivation of the risk estimates from the BEIR-3 report is presented in Appendix G. A brief outline of the general procedure is summarized below. Tables referenced from Chapter V of NAS80 are designated by a V prefix.

(1) The total number of premature cancer fatalities from lifetime exposure to 1 rad per year of low-LET radiation is constrained to be equal to the arithmetic average (280 per million person rad) of the absolute and relative risk values (158 and 403) given in Table V-25 of the BEIR-3 report (NAS80) for the L-L and $\overline{\text{L-L}}$ models for leukemia and solid cancers, respectively.

(2) For cancers other than leukemia and bone cancer, the age and sex-specific incidence estimates given in Table V-14 were multiplied by the mortality/incidence ratios of Table V-15 and processed through the life table code at constant, lifetime dose rates of 1 rad per year. The resulting deaths are averaged, using the male/female birth ratio, and proportioned for deaths due to cancer in a specific organ as described in Appendix G. These proportional risks are then used to allocate the organ risks among the (235.5) deaths per million person rad remaining after the 44.5 leukemia and bone cancer fatalities (Table V-17) are subtracted from the arithmetic average of 280 given in Table V-25.

(3) The RADRISK code calculates dose rates for high- and low-LET radiations independently. A quality factor of 20 has been applied to all alpha doses (ICRP77) to obtain the organ dose equivalent rates in rem per year. The derivation of the proportional organ risks and mortality coefficients for alpha particles are, however, based on the dose in rad as described in Appendix G, Table G-6.

A typical environmental analysis requires that a large number of radionuclides and multiple exposure modes be considered. The RADRISK code has been used to obtain estimates of cancer risk for intakes of approximately 200 radionuclides and external exposures by approximately 500 radionuclides. For each radionuclide and exposure mode, we assume that each member of a cohort of 100,000 persons is exposed to a constant radionuclide intake of 1 pCi/year, or a concentration of 1 pCi/cc-year for air immersion, or of 1 pCi/cm^2-year from the ground surface, until they die or are 110 years old, the maximum cohort. The mean life span of the cohort population is 70.7 years, a result obtained from 1970 age-specific mortality rates. The calculated dose rates and mortality coefficients described in the preceding sections are then processed through the life table subroutine of the RADRISK code to obtain lifetime risk estimates. At the low levels of contamination normally encountered in the environment, the life table population is not appreciably perturbed by the excess radiation deaths calculated and, since both the dose and risk models are linear, these unit exposure results may be scaled to reflect excess cancers due to the radionuclide concentrations predicted in the analysis of a specific source.

As noted in the discussion of the life table analysis, risk estimates for chronic irradiation of the cohort may also be applied to a stationary population having the same age-specific mortality rates as the 1970 U.S. population. That is, since the stationary population is formed by superposition of all age groups in the cohort, each age group corresponds to a segment of the stationary population with the total population equal to the sum of all the age groups. Therefore, the number of excess fatal cancers calculated for lifetime exposure of the cohort at a constant *dose rate* would be numerically equal to that calculated for the stationary population exposed to an annual *dose* of the same magnitude. Thus, the risk estimates may be reported as a lifetime risk (the cohort interpretation) or as the risk ensuing from an annual exposure to the stationary population. This equivalence is particularly useful in analyzing acute population exposures. For example, estimates for a stationary population exposed to annual doses which vary from year to year may be obtained by summing the results of a series of cohort calculations at various annual dose rates.

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